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DEVOTED TO DISEASES OF THE

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ORALISM AND AURALISM

A QUARTERLY JOURNAL DEVOTED EXCLUSIVELY TO PROBLEMS OF THE

DEAF AND DEFECTIVE SPEECH

EDITOR

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St. Louis

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THE
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Vol. XXXIII ST. LOUIS, JULY, 1923 No. 7

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

CLINICAL STUDIES OF VESTIBULAR AND AUDITORY TESTS IN INTRACRANIAL SURGERY.

DR. WELLS P. EAGLETON, Newark, N. J.

Chiefly with reference to the importance (1) of the absence of reactibility of the vertical canals of both ears to stimulation by the cold caloric *in the diagnosis of increased intracranial pressure of the posterior fossa,* and (2) of the reduction of the duration of the nystagmus from turning in diffuse lesions of the cerebro-spinal system pathways over the cerebellar cortex (as in protective meningitis and linear fractures of the skull of the posterior fossa), with a description of a new vestibular symptom complex.

VESTIBULAR REACTIONS.

SYNOPSIS.

I. Technic:

A department of neuro-otology under the direction of an otologist but largely operated by a trained technician, is advised in all hospitals.

II. Auditory Apparatus:

(*a*) *During the evolution of life the auditory apparatus was a comparatively late development; it is a very highly specialized function.*

(*b*) *The accepted hearing tests are largely valueless for differential diagnostic purposes as far as the central portion of the auditory apparatus is concerned.*

Presented in parts before the American Laryngological, Rhinological and Otological Society at its annual meeting at Atlantic City, June 1, 1921; The American Clinical College of Surgeons in Philadelphia, October, 1921; The New England Oto-Laryngological Society, Boston, March 30, 1922; and the American Laryngological, Rhinological and Otological Society, Washington, May 6, 1922.

III. Vestibular Apparatus—Biological:

A vestibular apparatus for orientation is well developed in very low forms of invertebrate life; it is one of the earliest of the special senses. Because of its early development it has become largely an inherent property of nervous tissue. It is doubtful whether any one special central tract is indispensable to the performance of orientation. Consequently, injury to or destruction of a vestibular tract or pathway is quickly and at times completely compensated for.

Clinical Application:

Because of these biological facts a fundamental mistake has been made in trying to formulate rules only applicable when dealing with highly specialized tracts such as are present in the speech mechanism.

Clinico-physiological:

(1) Because of the low and primarily reflex (automatic) activity of the vestibular mechanism (within the cerebellum), compensation is easily established; consequently, gross surgical lesions affecting the central vestibular apparatus, if of slow growth, may produce either very transient spontaneous symptoms, or in chronic lesions, no symptoms whatever.

(2) For the same biological reason, in interpreting the findings of the vestibular tests, the presence or absence of the following factors should always be considered, as they modify the vestibular manifestations:

(a) Is the lesion microscopic or diffuse?

(b) Has compensation had time to be established? and, consequently, is the lesion acute or chronic?

(c) Is increased intracranial pressure present?

(3) In microscopic lesions acutely destructive of a so-called "vestibular pathway" not associated with increased intracranial pressure, exact localization by means of the vestibular manifestations alone frequently is possible; as in minute hemorrhages, disseminated sclerosis and syringomyelitis.

(4) The vestibular manifestations of increased intracranial pressure, viz., the reduction of the reactibility of the vertical canals of both ears to the cold caloric (the reduction of the opposite side, in the author's opinion, being the greater) is of the greatest clinical value.

(5) In surgical lesions, because of the rapid and more or less complete establishment of compensation and the over-shadowing vestibular manifestations of increased intracranial pressure, locali-

zation of lesions by an examination of the vestibular apparatus alone is difficult, and generally impossible.

(6) Certain localized cerebellar surgical lesions, such as acoustic tumors, frequently can be diagnosed by the vestibular tests alone; the vestibular manifestations in such cases being a combination (1) of the effect of direct pressure on the vestibular tracts between the labyrinth and the brain stem—viz., the homolateral deafness, the absence of reactibility of all the homolateral canals, the spontaneous falling, and (2) of the effect of the increased intracranial pressure of the posterior fossa in abolishing the reactibility of the vertical canal of the opposite side.

(7) In surgical intracerebellar lesions, such as gliomas or diffuse suppurative lesions and abscesses, localizing evidence may or may not be obtainable from the vestibular reactions; but findings frequently are so transient and changeable, and as yet so imperfectly understood, that localizing surgical diagnosis can rarely be fundamentally based upon the findings of the vestibular tests alone.

(8) However, at any moment, due to an invasion of an intracerebral portion of the vestibular apparatus, spontaneous or induced, vestibular manifestations may appear, of localizing value—spontaneous vertical nystagmus, lateral deviation of the eyes—all of which usually rapidly disappear because of compensation.

(9) In exceptional cases the spontaneous vestibular manifestations continue for indefinite causes probably due to invasion of but without destruction of the vestibular nuclei in the brain stem.

Clinical Appliciation:

The immediate reduction of the duration of the nystagmus obtainable from stimulating the horizontal semi-circular canals by turning, is of distinct surgical and medico-legal value in diagnosing certain diffuse lesions of the posterior fossa. In the author's experience, this occurs in those lesions which are associated with a blockage of the cerebro-spinal fluid pathways by exudate, such as in protective meningitis or by blood in fracture of the skull, chiefly of the posterior fossa.

Conclusions:

(10) These two vestibular manifestations, viz., of increased intracranial pressure of the posterior fossa, and of blockage of the cerebro-spinal fluid system pathways of the posterior fossa, should always be considered in all attempts at localization of surgical lesions of the posterior fossa. In the author's experience they are the two chief intracranial vestibular manifestations so constantly pres-

ent as to be of consistent practical diagnostic value in cerebral surgery.

There is clinical evidence that sudden invasion of the frontal lobe causes spontaneous past-pointing. This rapidly disappears. There is also clinical evidence that too sudden invasion of the temporal lobe causes loss of quick component and induced nystagmus by application of the cold caloric applied to the side of the lesion. This also rapidly disappears.

There is clinical evidence that a sudden involvement of the cerebellum will cause nystagmus or a lateral deviation of the eyes. Both of these also rapidly disappear although in certain lesions, such as tumor and irreparable damage probably to the cerebellar nuclei, the nystagmus persists.

VESTIBULAR AND AUDITORY TESTS IN INTRACRANIAL SURGERY.

I. Technic of Otological Examinations in General.

At present the routine aural examinations in our large hospitals, if conducted at all, consist of an inspection of the drum membrane and a test to ascertain if the patient hears with each ear separately. Yet in every general hospital there is constantly a mass of material which, if a systematic effort was made to correlate it, would throw light on this complicated subject.

In my opinion, all cases that enter the surgical wards for cranial injuries should have during treatment, or at least before leaving the hospital, a routine otological examination by the functional tests. The same should apply to all cases in the medical wards which complain of vertigo, tinnitis or deafness; while, of course, no neurological examination should be regarded as complete without containing a full record of the vestibular reactions, both spontaneous and induced.

To make this possible there should be established in all general hospitals a department in charge of an otologist, although in my opinion the technical part of the work should not be required of him personally. All routine technical details should be delegated to a male or female technician of intelligence. It is asking too much of the otologist to require him to devote the large amount of time necessary for the tests, with—in the majority of cases—negative findings. It would be as reasonable to ask the attending physician or surgeon to perform the routine urine examination.

The technician should be instructed by the otologist how to conduct the tuning fork tests; how to measure the duration of the bone conductions and to perform calorics on the vertical and horizontal

canals. His report should be turned over to the otologist, and from it the otologist should make his diagnosis, verifying all abnormal findings. A few hours devoted to the instruction of such a technician in the use of the turning chair and the cold caloric—at first on a series of normal cases, followed later by abnormal ones—would soon make this possible. For the chief requisite is not medical knowledge, but keen powers of observation, associated with patience in execution, truthfulness in recording, and enthusiasm for the work. Until this division of the technical work is generally established, we shall make little progress in the vestibular tests.

The above is put forward, not as a suggestion but as a special plea, because work so conducted has been admirably successful in

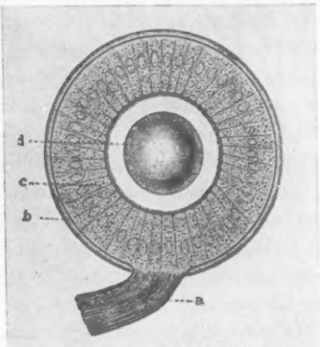

FIG. 240.—Otocyst of Unio.
a, acoustic nerve; *b*, capsule of the otocyst in which it ramifies; *c*, vibratile epithelium; *d*, otolith.

Figure 1. Vestibular Apparatus in Bivalve (River Mussel) Showing Complete Vestibular but no Auditory Apparatus.
From Morat, J. P.: Physiology of the Nervous System. Keener, Chicago. 1906.

the two instances in which I have had authority to enforce it.

II. VALUE OF AUDITION TESTS IN CEREBRAL LOCALIZATION.

Evolutionary Data of Clinical Value:

Audition is one of the late developmental additions in the evolutionary ascent of life. It is by means of the auditory function that speech was evolved, and from speech have come all the still higher psychic attributes possessed by man. Consequently, the auditory apparatus, both central and perpheral, is highly specialized—higher even than the visual apparatus. In man one of the evidences of this is that, although there is a hearing centre situated in both hemispheres, the auditory apparatus is the only one of the special senses,

outside of speech, which in certain parts *still has a tendency* to a cortical centre localized in but one cerebral hemisphere—the left; for while destruction of the hearing centre in the horizontal gyrus (Hershel's gyrus) of one temporo-sphenoidal lobe does not cause deafness, in certain cases injury to this gyrus on the left side will occasion word deafness.

All the other senses have in the evolution of life become represented equally in the brain cortex of both hemispheres.

An auditory apparatus is only present in the comparatively higher forms of life, in contradistinction to the vestibular apparatus which is well developed in very low types. While an otolite (with neuro-epithelial cells, the primitive part of the vestibular apparatus) is present in a very low form of invertebrate (in one of the low sub-divisions of the coelenterata), and the primitive medusa (jelly fish) has a well developed vestibular apparatus ("an otocystic formation with ciliated cells connected by nerves to a nerve ganglion, and this in turn with muscles"*), there is no evidence of a hearing apparatus in the invertebrate kingdom. It is only among the vertebrates in which the vestibular apparatus is fully developed, that a hearing apparatus first appears. From these rudimentary beginnings it is gradually developed up to and in the mammals and becomes complete in man. Fish have no cochlea, although possessing a well developed vestibular apparatus with a utricle and three-semi-circular canals. Snakes have a rudimentary cochlea (reduced to a quarter of a turn). "In birds the cochlea generally remains rudimentary, but the semi-circular canals are highly developed."*

This comparatively late acquisition by animal life of this highly specialized sense, audition, explains the clinical facts:

(1) that on account of its high specialization slight injuries are apt to destroy it totally;

(2) that injuries to the apparatus are slowly or never compensated for;

(3) that it is also because of its high specialization that we have as yet no proper diagnostic means of *localizing central lesions of the auditory apparatus* outside of the clinical appearance of word deafness.

We must realize that hearing is not only a highly specialized function, but it is an intellectual—psychic—function above the reflex-subconscious sphere, in which the vestibular function fundamentally resides. For to hear and interpret, we must listen.

*Morat, J. P.: Physiology of the Nervous System. English Edition, 1906, p. 601.

Audition, however, has an intimate association with the subconscious.

A cat, after the removal of the cerebral hemispheres, striata and thalami, shows orientated movements of the pinna and neck in response to sounds, such as a bark, whistling, et cetera. A protective reaction by the auditory motor is the jump reflex—showing how low down in the subconsciousness is the association between the reaction to sounds and orientation, the chief end-organ of the latter being the vestibular apparatus.* But the decerebrated cat does not hear in the higher sense in which the patient values hearing, for the purpose of intercourse with his fellows.

To hear speech we must listen, and this necessitates a motor and an intellectual effort. For if we do not listen, in church, for instance, the speaker's voice soon fades away as well as the text about which he is discoursing.*

TESTS FOR LOCALIZING LESIONS OF THE AUDITORY APPARATUS.

Although the violin for a musical instrument is the nearest approach to the human voice, in reality it bears no direct relation to it; in fact, no musical instrument can be adapted to act as a standard for speech, for the auditory apparatus of man, both peripheral and central, is especially specialized to perceive and interpret the human voice. From the viewpoint of the patient and from a proper diagnostic standpoint, it matters little how well or how poorly musical tones are heard if the modulations of the human voice are not correctly perceived.

But by some mischance the tuning fork, which furnishes a standard of *musical* tones, was adopted to differentiate the extent and position of lesions of the auditory apparatus.

It will require a much more delicate apparatus, based on a totally different principle, before we shall be able to diagnose the exact location of the lesions in the central portion of the auditory apparatus; for speech is made up of considerably more than a narrow range of musical notes.

Of the large number of musicl instruments that have been put on the market as aids to *differential diagnosis of the site of the lesion, whether peripheral or central,* I should say (from a long personal experience with the following—Hartman's Set of Tuning Forks, Edelman's Forks, Gradenigo's Visual Forks, Koenig's Rods, Polizer and Hay's Acumeters, and Galton's Whistle) that they are all *largely* overrated. Whatever practical information may be useful in differentiation is obtainable from one fork of long

*Sherrington, C. S. and Forbes, A.: American Journal of Physiology, 1914, Vol. XXV, p. 367.

*"The most satisfactory theory is that in the act of listening the resistance at the various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrites, which brings those of contiguous neurones into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the retraction of the dendrites." Hurst, Arthur F.: Psycology of the Special Senses—Hearing, Listening, and Hysterical Deafness. The Lancet, August 7, 1920, p. 285.

duration, excepting possibly in the case of partially deaf mutes, with whom my experience is so limited that I am not qualified to speak.

On account of the great difference in the personal equation between different patients and observers, it is necessary that a tuning fork over 200 d. v. per sec. be used uniformly to obtain even the meagre diagnostic information that the tuning fork may furnish.

III. The Practical Diagnostic Value—General and Localizing of the Vestibular Reactions in Cerebral Surgery.*

Present State of Knowledge:

The vestibular reactions of the end-organ—labyrinth—involvement are now fairly well understood, although many of the views of the physiology and pathology of the labyrinth itself are entirely hypothetical and are being constantly modified.

The anatomical and physiological knowledge of the cerebral portion of the vestibular apparatus is, however, still very imperfect.

Following the initial and enthusiastic acceptance of the localizing assistance of the vestibular tests in intracranial lesions, their practical value is today being seriously questioned. We must frankly admit that the subject is still in considerable confusion, and many of the findings are as yet unexplainable.

Disastrous Results of Hypothetical Teaching, not Based on Clinical or Experimental Experience:

An injury has been done and a proper study of the tests retarded by the publication as facts of what in reality are but hypothetical deductions. Undue emphasis has been placed upon selected and isolated cases in which microscopic lesions of the cerebellar peduncles and of the posterior longitudinal bundle have been successfully diagnosed.

The positiveness of the statement created the impression that the tests, if properly performed and interpreted (from the viewpoint of adequate anatomical and physiological knowledge of the nervous system) are so accurate that a diagnosis of the localization of lesions of the medulla pons and parts of the cerebellum may be uniformly made (although anatomically a large part of these "tracts" still are problematical); the inference, of course, being that when such a diagnosis is not forthcoming the fault lies with the observer and not with the tests themselves.

There is no doubt that in very exceptional cases in microscopic lesions, such localizing diagnosis is possible; but the publication of a few isolated, brilliant diagnoses has discouraged the independent

*Read in part before the American Otological, Rhinological and Laryngological Society at its Annual Meeting at Atlantic City, June 1, 1921.

worker, because failing to obtain localizing assistance in the average *surgical* case, he naturally has regarded the fault as with himself—in his lack of ability to interpret the findings correctly. The effect has been to intimidate otologists, make skeptical the neurologists and neuro-surgeons, and, in consequence, to discourage a continuation of the work.

Fallacy of Attempting to Apply Theoretic Physiological Deduction to Surgical Lesions:

In those books in which few or no references to individual work are made, the brain is regarded as an inert body—a sort of complicated system of telephone wires, through which it is possible to prevent such and such a transmission "by interference" with such and such a tract without disturbing the other tracts; much as a lineman "cuts out" a wire.

It appeared so logical that interference with a pathway should cause loss of this or that part of a reaction that for a long time (in common with the rest of the profession) I failed to perceive the fundamental defect in the application of these theories to surgical diagnosis.

In the final analysis, all real scientific progress must rest on the accumulated experience of many. Theoretical considerations, which are not based on and fully verified by experience, tend, by misleading, to retard a proper understanding of disease. Some of these postulates are undoubtedly true, but as always occurs in hypotheses not founded on experience, in these publications (one copying from another) certain factors present in surgical lesions of the brain were disregarded, viz., increased intracranial pressure, the acuteness and nature of the lesion, and the influence of the establishment of compensation.

All these factors fundamentally modify the objective reactions of the functional action of the brain—which the vestibular reactions primarily are.

Prior to 1914, having devoted considerable attention to the vestibular tests and having become somewhat discouraged by the diversity of the findings, I was not only impressed by these published accounts as truths by the facility with which they apparently clarified so many complicated problems, but also convinced that my failures were due to my inadequate knowledge. Not realizing their fundamental weakness, I abandoned my preconceived opinions, the result of experience, and adopted the published statements as demonstrated facts, only to find after many years that the statements were in many cases fallacious.

This has been the general effect upon the profession, as an examination of the literature of the past few years will demonstrate; for not only has little been written on the subject, but what has appeared deals in generalities of the most elementary nature. More significant still is the fact that while the reports of cerebro-surgical cases of brain tumors have enormously increased, few of them contain detailed records of the vestibular reactions; which forces the inference that the vestibular tests in cerebro-surgical conditions either have been generally abandoned, or have been of so little assistance as not to warrant their incorporation in the published reports.

Physiological Considerations:

The vestibular reactions are, as we have seen, in many particulars very primitive reflexes of orientation and easily compensated for the reason that the more primitive the reflex the more diffuse and varied are the ways by which such impulses are transmitted, the reflex action being assumed by the nervous tissue as a whole. Thus, a man with a paralyzed arm from cortical destruction still gives a muscular twitch when a primitive protective reflex is stimulated, such as "auditory jump reflex," although the efferent tract necessary for voluntary movement is absent.

This probably explains why so many of the vestibular pathways have not been isolated, there being in fact no one definite bundle of fibres in many instances. In such a case all we are able to do is to study and attempt to interpret an absence or perversities of the whole or part of a normal reflex; and even in gross surgical lesions, in which large areas of cerebral tissue are affected, these perversities may not be present because of the establishment of compensation or because of the absence of definite cerebral localization of the function itself.

It is doubtful whether in very primitive reflexes any definite nerve tissue arc or pathway exists in the cerebral tissue, for we know that in spinal reflexes there is a "choice of efferent paths by which the excitation can be transmitted to the peripheral motor organ."

Clinical Method of Interpretation Necessary:

If the vestibular tests are to occupy a place of practical value in the differential diagnosis of surgical and non-surgical lesions, I believe it is necessary that the present method of their interpretation from theoretical generalizations, based on supposed "interpretation with this or that" so-called "vestibular pathways," must be largely abandoned, and the abnormal vestibular reactions listed as they are clinically present in different diseases and lesions. It is only by

Luciana: Human Physiology. Vol. III, p. 93.

some such method that definite information of clinical value can be obtained from the apparently confusing reactions.

In the present state of our knowledge regarding the nervous mechanisms whereby the vestibular reactions are transmitted, an attempt at cerebral localization based upon the supposition of "interference with vestibular pathways" is sure to end disastrously.

If, however, the abnormal vestibular reactions are especially listed under a *clinical classification* (of lesions and diseases), the vestibular manifestations being regarded as more or less clinical symptoms (of such lesions and diseases); such data in a large number of cases having been obtained it will then, and only then, become possible to argue backward, with the recognition of the well-known clinical lesion or disease as a basis, and to attempt exact cerebral localization by the assistance of the known vestibular manifestations.* It is only by such a slow, laborious and uninspiring method that real progress is possible.

Clinical Classifications of Lesions and Diseases in the Presence of Abnormal Vestibular Reactions Which May Occur as a Symptom:

(A) Non-surgical lesions which because of their more or less microscopic size are *not associated with increased intracranial pressure:*

(1) Chronic degenerative lesions; syringomyelia, disseminated sclerosis, locomotor ataxia, Friedreich's ataxia.

(2) Vascular disease; minute hemorrhages; small vascular occlusions.

(3) So-called functional nervous disease, such as chorea, epilepsy, etc.

(B) Surgical Lesions; chief among which, because of their constant presence in all surgical lesions, should be enumerated:

(1) The vestibular manifestations of increased intracranial pressure in general;

Having recognized that increased intracranial pressure, especially of the posterior fossa, gives rise to a distinct vestibular manifestation (which should be classed among the symptoms of increased intracranial pressure the same as papilloedema), we can then further sub-divide the vestibular manifestations of surgical disease according to the origin and the location:

(2) Localized suppurative diseases of the posterior fossa;

*Eagleton, W. P.: Brain Abscess—Its Surgical Pathology and Operative Technic—Macmillan, 1922. (In press.)

(a) Protective meningitis from labyrinth suppuration in intrapiarachnoid abscess,

(b) Intracerebellar abscess confined to the hemispheres of the cerebellum,

(c) Intracerebellar abscess invading the vermis.

(3) While the vestibular manifestations of *Cerebral Abscess* should be divided with those present into,

(a) Temporo-sphenoidal lobe abscess,

(b) Frontal lobe abscess,

(c) Occipital lobe abscess.

(4) *Surgical Non-suppurative Lesions* with increase of the intracranial pressure, do not lend themselves to analysis with the same facility as localized suppurative diseases not only because they have not the same tendency that a brain abscess has for the selection of definite sites at the beginning, except in a few particulars, but also because of the chronic nature of their invasion they are not apt to give the clean-cut picture of vestibular manifestations induced by a localized area of acute suppuration; for while tumors (encapsulated) are frequently more circumscribed than brain abscess, compensation has had greater facility for re-establishment. Consequently, it is essential that they be listed separately from suppurative diseases.

The vestibular manifestations of brain tumors should be listed under those present as follows:

(1) *Tumors below the tentorium:*

(a) Tumors of the acoustic nerve,

(b) Tumors of the pontine-cerebellar angle,

(c) Intercerebellar tumors of the cerebellar hemispheres,

(d) Tumors involving the vermis.

(2) *Tumors of the cerebrum:*

(a) Pituitary growths,

(b) Suprasellar growths,

(c) Pineal tumors,

(d) Frontal lobe tumors, Anterior to the Rholandic fissue,

(e) Temporo-sphenoidal lobe tumors,

(f) Motor-tract tumors, Post Rholandic,

(g) Occipital lobe tumors.

I am perfectly aware of the many weaknesses of such a classification, but as it is based upon the usual lesions and their sites most frequently selected, it has some practical value, especially as during the period of invasion and before the establishment of compensation the vestibular manifestations are apt to be the most clear-cut and, consequently, of the greatest assistance.

Accidents During Performance of Vestibular Tests:

In addition to the possible danger of causing a recurrence of a previous purulent otitis by the application of a cold caloric, it should not be denied that the rotation test may, under certain conditions, precipitate alarming or even dangerous disturbances. This has occurred to me in two instances.

CASE REPORTS

Case I: A girl of twelve years, prior to the evacuation of a temporosphenoidal lobe abscess, had a convulsion. The operation was followed by a small hernia which soon became completely covered with skin, and the patient was discharged as cured. When she was recalled for examination three years later, there was a slight bulging about the size of half a marble over the site of the old hernia. The girl apparently had been in good health for three years. In the midst of the rotation she had a convulsion, became unconscious, and apparently was dying. From this state she gradually recovered and was able to be returned to her home about two hours later. When it is recalled that for three years she had had no cerebral symptoms, and that the only previous convulsion undoubtedly was due to her brain abscess, the inference is forced upon us that the rotation caused a severe disturbance, probably vascular, of her brain—made possible, perhaps, by the dural defect.

Case II: A woman, in her fifties, with a high blood pressure, developed a slight exophthalmos, which appeared first in one eye and then slightly in the other. At the time of the examination the veins of both ocular conjunctiva, especially of the left, were distended, as were the veins in both fundi, in which there were multiple small hemorrhages. R. V. 20/50; L. V. 20/70. There was a paralysis of the left external rectus. A distinct bruit could be heard all over the head, chiefly on the left side, which she said had been present on and off for the previous eighteen months, coming on suddenly and then gradually subsiding. (This and the diminished hearing in both ears, but especially in the right—the contralateral side—had been attributed to her high blood pressure). The Weber was referred to the right. She gave a history of severe headache, accompanied by vomiting and swelling of her lids.

A diagnosis of pulsating exophthalmos from aneurismal varix of the internal carotid, with rupture into the cavernous sinus of the left side, was made. During her neurological examination it was discovered that she had a *spontaneous deviation* of the arm of *the contra-lateral side inward,* about two inches. I ordered that she be rotated. Rotation to the right; horizontal nystagmus, 24 seconds; no vertigo, but she vomited; rotation to the left; horizontal nystagmus, 23 seconds; no vertigo. Immediately following the rotation she had a severe pain in her head and began to vomit; both eyes protruded greatly, associated with such an intense oedema of the skin that the eyes could not be opened, and there were numerous fresh hemorrhages in both retina. On the following day she was able to recognize hand movements only in one eye, the other being totally blind except to light.

This attack resembled her previous ones, but was much exaggerated. For several days I feared she would die. Remarkable to relate, during this time

the pulsation in her head entirely disappeared, both subjectively and on auscultation, and never recurred.

Four days after the rotation I ligated the left common carotid artery in the neck. This was promptly followed by a recession of the eyeballs, and later by absorption of the hemorrhages. One month later, R. V. 20/60; L. V. 6/200. The vision of the right eye—the contra-lateral eye—now began to fail; the conjunctival veins enlarged and fresh hemorrhages appeared. The hemorrhages were associated with an attack of glaucoma of the right eye, which rapidly caused her to become blind in that eye, while in the left—the primarily affected eye—the vision improved and today she has a vision in her left eye of 20/30.

As in the previous case, it undoubtedly was an abnormal cerebral vascular system which caused the accident. There is, of course, no excuse for the latter case; she should not have been subjected to rotation nor a caloric, but with the former it shows that in vascular disturbances, either pathological or traumatic, rotation is not without some risk.

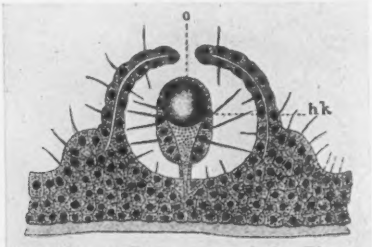

FIG. 239.—Auditory organ of the Rhopalonema still showing a small orifice (Hertwig).

hk, modified tentacle; *o*, auditory organ.

Figure 2. Vestibular Apparatus In Butterfly Showing Highly Developed Vestibular Apparatus But Absence of Auditory Apparatus. From Morat, J. P.: Physiology of Nervous System. Keener, Chicago, 1906.

Biological Data of Clinical Significance About the Vestibular Apparatus:

Even in very low forms of life a well-developed vestibular apparatus is present, because orientation is necessary. In the invertebrate life very early evolved a vestibular apparatus out of one of its most primitive attributes—viz., sensation; for "functionally, the vestibular apparatus is a transformation of the tactile apparatus adapted to the analysis of pressure." *Fig. 2. It was not until much later, as we have seen, that life in the verebrate evolved an auditory apparatus for the further analysis of the waves of sound.

This fundamental evolutionary fact that the vestibular apparatus is of primitive origin, well developed in the invertebrate, and audi-

*Morat, J. P.: Physiology of the Nervous System, 1906, p. 602.

tion is of much later development, first appearing among the vertebrates, and then very rudimentarily, is of prime importance; for the simpler the form of life, the more easily is it able to dispense with its parts, or rapidly compensate for the loss of them or injury to them. This explains the ability possessed by man of rapidly compensating for or entirely dispensing with this rudimentary apparatus. It furnishes the clue to the clinical facts that the loss of or injury to the vestibular apparatus, both peripheral and central, is rapidly and even completely recovered from by the establishment of compensation, while damage to the auditory apparatus is permanent, there being no compensatory machinery to take its place. A man loses the hearing of both ears from suppuration—his vertigo, nystagmus and spontaneous pointing deviations soon disappear, but his hearing never returns.

During the past few years, as the wonders of recent discoveries regarding the vestibular apparatus have dazzled our intellect and stimulated our imagination, we have regarded the vestibular apparatus as highly specialized, on the same plane with speech, which possesses very definite "tracts", and an injury to which produces definite and irreparable manifestations. In this our error has originated. Because the vestibular apparatus is so *new to us,* we have treated it as new to life, while in reality to life it is one of the oldest attributes—not "as old as the hills," but much older; for when life acquired it the waters covered the surface of the earth, and the hills were not yet.

A. Vestibular Tests in Non-surgical Lesions.

In a consideration of vestibular reactions the presentation of a clear-cut picture—a loss of such parts of otherwise normal vestibular reactions sufficient to warrant a vestibular peduncular symptom complex or a vestibular posterior longitudinal symptom complex—must presuppose a very small lesion, so small, in fact, as to be almost microscopic. In multiple sclerosis, syringomyelia and Friedreich's ataxia the vestibular reactions are of the greatest localizing value because the lesions are degenerative and microscopic in extent, without a complicating increase of intracranial pressure. Only in such lesions will the rules formulated for "interference with such and such a vestibular pathway" find a proper application.

B. Vestibular Tests in Surgical Cranial Lesions.

Visualization of the Lesion—Not "Interference With a Vestibular Pathway"—Necessary for Surgical Localization:

In considering the vestibular reactions in their relation to surgical lesions of the brain, the surgeon should not regard the intracranial

portion of the vestibular as definite inflexible tracts, which are either opened or closed as an electric circuit. Unfortunately, surgical cerebral pathology is not so simple.

The surgeon must think of something which can be definitely visualized—an isolated area, such as an endothelioma, cyst, or extradural hemorrhage, or a portion of the brain surrounded by a zone of induration, such as an abscess or an infiltrating glioma.

All surgical lesions cause an increase in the intracranial contents and give rise to profound disturbances in all parts of the intradural tissues, influencing the vestibular reactions along with the rest, although the growth or abscess may not be situated directly in the course of any vestibular neuron. Consequently, when we attempt to apply the rules of vestibular tract localization (which from a pathological standpoint may be accurate), the surgeons must not leave out of consideration the vestibular manifestations of the increased intracranial pressure itself, which not only modify but frequently overshadow the other vestibular manifestations.

Factors Infuencing and Altering the Vestibular reactions in Surgical Lesions:

I. Increased intracranial pressure.

II. Establishment of compensation in the two vestibular apparati.

Corollary Factors:

III. Duration of the lesion—whether acute or chronic.

IV. Nature of the lesion—whether causing symptoms (a) by pressure alone, as in endothelioma, cysts, intradural or intrapial hemorrhages, or (b) by destruction or infiltration, abscess or infiltrating tumors.

1. *The Vestibular Manifestations of Increased Intracranial Pressure:*

Through ignorance of the work of other observers, the vestibular manifestations of increased intracranial pressure have been largely disregarded. *The vestibular manifestations of increased pressure, especially of the posterior fossa, are definite,* however, and so outspoken as to overshadow, and in the majority of cases, to prevent the localization of surgical lesions by the vestibular tests alone, even when an intracranial portion of the vestibular apparatus itself is directly involved, as in many cases of large cerebellar abscess; for *in surgical lesions* it must be realized that *the vestibular manifestations which stand out* most prominently *are the manifestations of increased intracranial pressure,* and these vestibular manifes-

tations are disclosed only by the caloric and turning tests, just as the ocular manifestation of increased intracranial pressure—papilloedema—is disclosed only by the ophthalmoscope.

A large personal experience would lead me to postulate that in the presence of increased intracranial pressure the vestibular reactions are of *great general diagnostic value, although they are but rarely useful in cerebral localization.* In my opinion, they should take, like the demonstration of a papilloedema by the ophthalmoscope, their position as of great clinical assistance in the diagnosis of increased intracranial pressure, especially of the posterior fossa.*

In the year 1912, in a communication to the American Otological Society*, after recording the history of three cases of tumor of the brain involving the posterior fossa and commenting upon *the absence of reaction to the cold caloric when applied to the ear of the opposite side of the lesion* in all three cases, I stated, "This condition, if generally present, may be of service in *diagnosing increased intracranial pressure* involving the auditory apparatus, possibly at an early stage", and I concluded with the statement, "A study of these three cases leads me to believe that in *intracranial lesions of the posterior fossa there may occur early reduction of the excitability of the vestibular apparatus, not only of the affected side but of the unaffected side as well.*

Prior to the appearance of my paper there was no recorded similar observation, although Neumann previously had called attention to the reduced irritability to the cold caloric of the contra-lateral side in suppurative otitis. Consequently, it is claimed that my observation was the *first recorded vestibular manifestations of increased intracranial pressure,* and more important still, that at that time *the first proper interpretation* was offered.

Since 1912 the accuracy of this vestibular manifestation of increased intracranial pressure has been substantiated in so many instances, both clinically and at post-mortems, that I have come to regard it as of the first importance. In my opinion, it is the failure to appreciate its uniformly overshadowing presence that accounts for many of the disappointments and for much of the skepticism in regard to the value of the reactions in surgical lesions.

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*It is strange that this error has been made, because of the former fallacy regarding the localizing importance of papilloedema.

*NOTE: Prior to the publication of this paper I had applied the cold caloric in the upright position, thus stimulating the vertical alone. I had no knowledge that in cases of increased intracranial pressure of the posterior fossa, a vestibular reaction follows when the opposite horizontal canal is stimulated.

Eagleton, W. P.: Recompression for the Relief of Disturbances of the Auditory Apparatus of intracranial Origin; Report of Three Cases with a Previously Undescribed Aural Condition. Transactions of the American Otological Society, June 10-11, 1912, Vol. XIII, Part III, p. 564. Also Laryngoscope, 1913, Vol. XXII, p. 592.

Because of its great importance to the clinician credit for whatever value the description and interpretation of the reactions possess is due to this early observation.

The vestibular manifestation of increased intracranial pressure, viz., the absence of reactibility to the cold caloric on the opposite side was frequently mentioned in the cases reported by other observers, but its diagnostic value apparently has not been appreciated. During the past few years this vestibular manifestation of increased intracranial pressure has been elicited by me in a large number of cases of various intracranial pathological processes. In numerous instances its reliability has been proved by a lumbar puncture; and on several occasions an amber colored fluid under pressure—showing a pathological condition—has been encountered in cases in which the routine otological examination had led me to believe a peripheral lesion was present.

I can confidently affirm that the demonstration of an increased intracranial pressure by the caloric has largely revolutionized my conception of many of the poorly understood intracranial lesions, such as frequent cases of deafness, psychoses, and headaches following trauma, and in several instances as well has rendered possible a diagnosis of probable brain tumor long before the appearance of a papilloedema.

Case III. THE VESTIBULAR MANIFESTATION OF INCREASED INTRACRANIAL PRESSURE, DIAGNOSING A PROGRESSIVE DEAFNESS AS OF INTRACRANIAL ALTHOUGH CLINICALLY OF PERIPHERAL ORIGIN; LUMBAR PUNCTURE VERIFYING THE DIAGNOSIS.

P. G., Male, 29 years of age.

Outstanding Symptoms:

Increasing deafness in both ears, especially the right, during the past five years; tinnitis in both ears; severe headaches.

Family History: In regard to hearing, negative.

History:

No preceding illness. For six years he had had loud noises in his ears and has been growing deaf gradually in both ears, with headaches and slight dizziness on moving or turning his head. He never had pain in nor discharge from his ears. The condition seems to grow worse each winter but remains stationary in the summer. He hears better in quiet surroundings and on clear days. The tinnitis in the right ear resembles escaping steam; in the left, a booming sound. The patient believes the deafness was noticed before the tinnitis. One year ago his tonsils were removed; within the last year he has had two operations on his nose—all to improve his hearing but without benefit.

Examination: O. E. negative. Vision normal with proper correction. *Mouth:* Teeth showed the gums of upper jaw red high up near the fold of the membrane. X-ray of teeth, negative. *Nose:* The left inferior turbi-

nate seemed to run far back and hang low posteriorly; membrane glossy; no pus. Transillumination showed no definite shadow but was not bright on either side.

Blood pressure, 128 over 90.

Hearing: Watch heard at one foot; whisper at five feet; voice across room. Both canals were negative; right drum negative; left slightly retracted.

Right Ear	A. C.	40 seconds	B. C.	110 seconds
Left Ear	A. C.	65 seconds	B. C.	115 seconds

Weber—Vertex—Sound referred alike to each ear.
Teeth—Sound referred to left ear.
Chin—Sound referred to left ear.

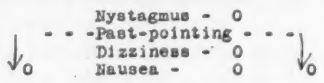
Rinne—Vertex—Sound referred alike to each ear.
Teeth—Sound referred alike to each ear.
Chin—Sound referred to left ear.

Vestibular Reactions:

R

Chart I. P.G.

Spontaneous



Rotating to Right

Nystagmus - ⟶ 46 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

Rotating to Left

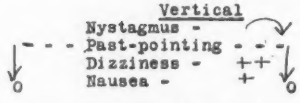
Nystagmus - ⟵ 44 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

October 11:

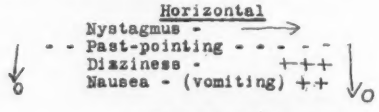
R

Cold Caloric in Right

Vertical

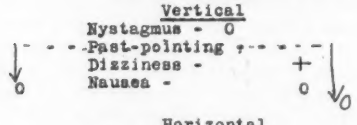


Horizontal

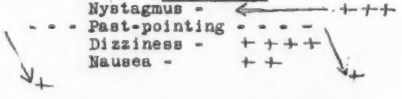


Cold Caloric in Left

Vertical



Horizontal



Blood Wassermann—Negative.
Blood Pressure—128 over 90.
Urine—Negative.

Lumbar Puncture gave fluid under very great pressure, opaque, of a slightly amber color; globulin and sugar, normal; cell count, 6.5 per cmm.

Abnormal Findings: Exaggerated patella reflexes; great exaggeration of the duration of the nystagmus; lack of proper pointing deviation; positive Rhomberg. "Stimulation of left vertical canal with cold caloric gave no nystagmus, some dizziness, no past-pointing; consequently, the trouble probably is a right-sided lesion."

Diagnosis: "In view of all the symptoms it would seem possible that the man has a disseminated sclerosis. It sounds like a toxic lesion; possibly a thyroid case. The amber-colored cerebro-spinal fluid is diagnostic of a degenerative lesion."

January 28:

Vestibular Reactions:

Spontaneous

Nystagmus - 0
Past-pointing - - -
Dizziness - 0
Nausea - 0
0
++++ (4 inches.)

Rotating to Right

Nystagmus - ⟶ 24 seconds.
Past-pointing - - -
Dizziness - ++
Nausea - 0
0 (See Spontaneous)
++++ (4 inches.)

Rotating to Left

Nystagmus - ⟵ 25 seconds.
Past-pointing - - -
Dizziness - ++
Nausea - 0
+ (Sligh 1 inch)
(See Spontaneous)
0

Increased intracranial pressure a handicap in localization by means of the vestibular reactions.

Surgeons and clinicians should appreciate that while in a microscopic lesion involving the vestibular tract, exact localization by the vestibular reactions of the site of the lesion either in the cerebellum, the cerebellar peduncles, the posterior longitudinal bundle, and even in certain parts of the cerebrum, is frequently possible; the presence of increased intracranial pressure of the posterior fossa, causing as it does a uniform absence or reduction of irritability of the vertical

canals of the opposite side as well as those of the affected side, generally prevents accurate localization by the vestibular tests alone.

Much in the same way an intense papilloedema, by causing a general contraction of the visual field, may obscure a localizing hemianoptic defect (in the visual fields).

In cases of increased intracranial pressure, the reduction of the irritability of the vertical canals of the opposite as well as those of the affected side renders a localizing diagnosis impossible by the vestibular manifestation alone.

II. Establishment of compensation in the vestibular mechanism—cerebral reduction of the duration of the induced nystagmus on turning:

Another factor of fundamental importance, which further interferes with the localizing value of the vestibular reactions and greatly complicates their interpretation in lesions affecting the intracranial portions of the vestibular apparatus, both gross and microscopic, is the early establishment of compensation. I also called attention to this in the paper before referred to, as shown in the reduction of the duration of an induced nystagmus and of the caloric excitability of the tracts of the two sides. This readjustment, to which I gave the name of "establishment of compensation", is intimately associated with and allied to the vestibular manifestations of increased intracranial pressure.

It may be imperfectly illustrated as follows: with a normal vestibular apparatus—end-organ and central—stimulation of a horizontal canal by turning produces an induced horizontal nystagmus of 24 seconds' duration, 16 seconds being contributed, it is believed, by the horizontal canal of the labyrinth chiefly stimulated and 8 seconds by the opposite labyrinth.

If one labyrinth is destroyed the maximum duration of induced nystagmus obtainable is 16 seconds, wholly contributed of course by the remaining intact labyrinth, while 8 seconds of nystagmus occur on turning in the opposite direction, this 8 seconds also coming from the remaining labyrinth.

In time, however, the duration of the nystagmus is again equalized—although one labyrinth continues out of commission—by, I believe, the establishment of compensation. The duration of the induced nystagmus on maximum stimulation by turning toward the destroyed labyrinth, which immediately after the labyrinthine destruction was 16 seconds, is reduced to 10 or 11 seconds; and the stimulation—on turning away from the lesion—which, immediately after the labyrinthine destruction had a duration of 8 seconds, now is increased almost to the same as that recorded in turning in the opposite direction, viz., to 10 or 11 seconds. In other words, a compensation has been established by dividing the duration of the induced nystagmus equally between the two sides, although one end-organ is now contributing all the stimulation.

C. Vestibular Tests in Diffuse Suppurative and Hemorrhagic Disease of the Meshes of the Piarachnoid of the Posterior Fossa:

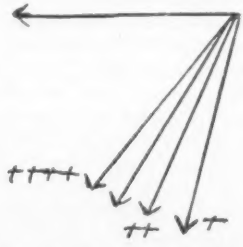
Even with these handicaps the vestibular tests, if routinely and conscientiously applied as part of the general neurological examination—just as the hearing and sight are tried and the eye grounds inspected—will be found to be of the greatest assistance and will, as our knowledge of the vestibular apparatus is amplified, become of more and more assistance in localization. Progress, however, can be made only by the publication of case reports, so that large numbers of detailed reports may be collected and properly analyzed.*

D. Vestibular Tests in Protective Meningitis of the Posterior Fossa from Labyrinth Disease or Intrapiarachnoid Abscess:

With this in view the following four cases are presented in detail. From them certain deductions are to be drawn. Whether, however, these deductions will be substantiated must depend upon the observations of many independent investigators.

Report of Four Cases of Protective Meningitis Following Labyrinth Suppuration or Trauma, Probably Associated with a Localized Collection of Cerebro-spinal Fluid Over That

*In the publication of reports of vestibular findings, all details must be incorporated, as the lack of minute details—such as omitting to state the exact position of the patient, whether lying in bed or sitting with his forward at 30° during the application of the cold caloric—may invalidate their value.



For accurate recording of details I would advocate following routinely the scheme of vestibular portioning and special vestibular tests suggested in the Guide to a Detailed Neurological Examination in "Fracture of the Skull with Chart of Clinical Classification and Guide too Detailed Neurological Examination," Eagleton, W. P., Archives of Surgery, July, 1921, Vol. III, pp. 140-153, in which an attempt is made to compel attention to details both during the examination and when recording it, while for purposes of rapid and visualized comparison, such as induced or spontaneous past-pointing and nystagmus, the author offers the visualizing charts given with the case reports in the present paper.

For the past-pointing deviations the following graphic scheme may be used:

Each X represents one inch until four XXXX, after which the angle should be used.

PORTION OF THE ANTERIOR SURFACE OF THE CEREBELLUM WHICH LIES BEHIND THE POSTERIOR SURFACE OF THE PETROUS PYRAMID; WITH A SUGGESTED DIAGNOSTIC VESTIBULAR SYMPTOM COMPLEX—NAMELY, (1) ABSENCE OF THE COLD CALORIC OF THE VERTICAL CANAL OF THE UNAFFECTED SIDE (FROM INCREASED INTRACRNANIAL PRESSURE), AND (2) IMMEDIATE REDUCTION BY 50% OF THE MAXIMUM DURATION OF THE INDUCED NYSTAGMUS ON TURNING, (WHICH WOULD BE OBTAINABLE WERE THE LABYRINTH ALONE AFFECTED); WITH (3) EVIDENCE OF THE INVOLVEMENT OF A PAST-POINTING CENTRE IN THE CEREBELLAR CORTEX—AS THE SPONTANEOUS POINTING DEVIATIONS COULD NOT BE ALTERED IN DIRECTION UNDER ANY FORM OF STIMULATION.

Case IV. CASE OF SEROUS MENINGITIS WITH PAPILLOEDEMA, CLOUDY FLUID, HIGH CELL COUNT, FOLLOWING LABYRINTHITIS; AND SINUS THROMBOSIS WITH ABSCESS OF THE JUGULAR VEIN; ASSOCIATED WITH SPONTANEOUS PAST-POINTING DEVIATION OF BOTH ARMS AWAY FROM THE LESION, WHICH COULD NOT BE ALTERED BY STIMULATION, ABSENCE OF THE OPPOSITE VERTICAL CANAL TO COLD STIMULATION, ABSENCE OF OPPOSITE VERTICAL CANAL TO COLD STIMULATION FROM INCREASED INTRACRANIAL PRESSURE—AND IMMEDIATE REDUCTION OF DEVIATION OF NYSTAGMUS TO EIGHT SECONDS.

(Successive charts show the persistence of the pointing deviation until altered by the opposite ear becoming affected, when there was a slight alteration.)

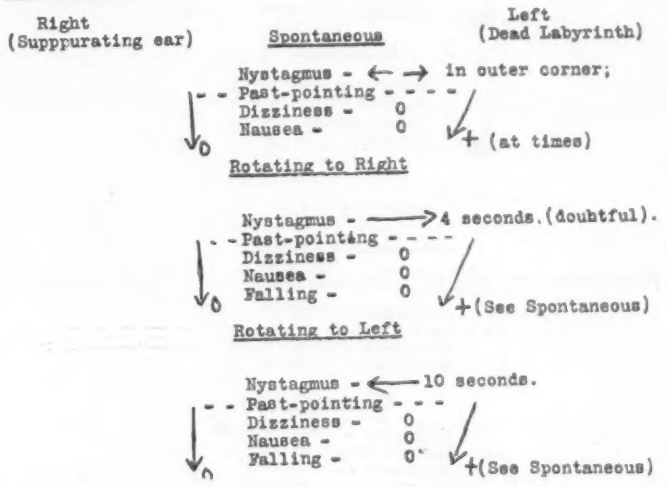
August 19, 1919, J. B., man, twenty years of age.

History: Has always had running left ear with frequent attacks of pain. Always has been subject to headaches. Four weeks previously had pain in his ear—a usual condition. The following day had a severe chill, followed by fever, then a sweat. This was repeated several times during the next four weeks, with considerable headache. Admitted to Newark Hospital. Examination disclosed chronic mastoid involvement. On opening the mastoid Dr. E. A. Curtis found a large septic clot in the sinus. Condition became very poor during the operation; man was thought to be dying. Returned to bed without jugular having been ligated. Did well for a few days; then there was a recurrence of chills and pain. About ten days later I exposed his jugular, found an abscess filling the whole of the interior of the vein itself. *Free pus in the vein.* It was not in the fascia surrounding it but in the vein. On slitting the vein, free pus was liberated—no clot. I followed downward and in the lower portion found a clot. Incision in vein went below the level of the clavicle but did not cause bleeding. Jugular bulb now exposed. Free bleeding was produced from the upper portion of the lateral sinus.

Examination of his eyes immediately prior to the second operation showed no change in the discs. Immediately after the second operation the man complained of dizziness, which contiuned. He felt well, but not so well as after the first operation. Continued to do fairly well, although he lost considerable flesh. Did not look well, although did not look sick enough to warrant any further interference.

Seven weeks after the original involvement, and ten days after the second operation, man had two chills, followed by a period of violent headaches, located in frontal region; headaches would pass away as suddenly as they came. Temperature not much elevated. Examination now revealed double papilloedema. (How long this had been present was not known. It was not present prior to the second operation.) His labyrinth now entirely dead. Had no spontaneous nystagmus, but a *spontaneous pointing deviation of both hands to the right* (away from the lesion). Cold caloric on left (affected) side produced no nystagmus, no vertigo—dead labyrinth; lost flesh. On walking, would fall to the right side (away from the lesion). Patella reflexes present. Had a slight area of anesthesia—only partial—around the third branch of the tri-facial. Had one severe attack of vomiting. Lumbar puncture revealed fluid cloudy, under pressure. Cell count, 160; increased globulin. Repeated blood cultures—all negative. No Romberg.

May 13, 1920:



Deductions from Examination: "It would seem from the pointing deviation to the right side and falling to the right side—both away from the lesion—that the inward pointing centre (Barany's) of the cerebellum opposite to the posterior surface of the petrous portion of the temporal is involved. This is also confirmed by the cerebro-spinal fluid, giving a protective meningitis with a slight area of anesthesia. If we rotate him and cannot produce a pointing deviation of the left, this may be confirmed."

Sept. 29, 1919. 1—Rotation to the right; no nystagmus; no dizziness; no nausea; no deviation of right arm; slight deviation of left to right. 2—Rotation to left; nystagmus to right, of eight seconds; very slight dizziness; no nausea. Deviation of right 2 inches to left; no deviation of left.

Third Operation: Incision of dura over anterior portion of cerebellum; evacuated considerable amount of turbid cerebro-spinal fluid; no pus; exploration of cerebellum, negative.

Uneventful recovery followed operation.

Chart I. J.B.

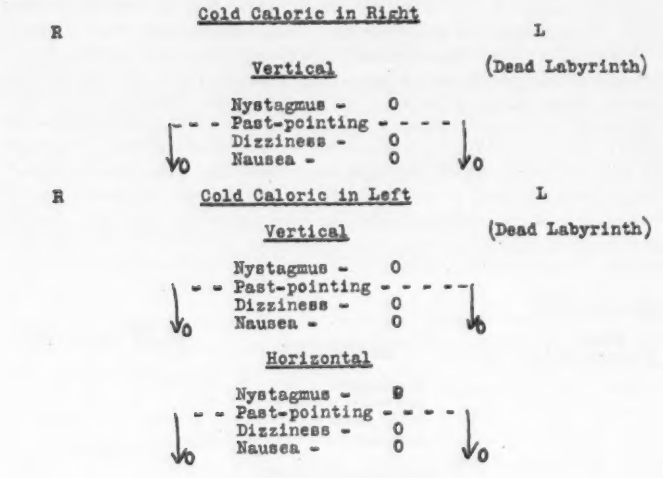
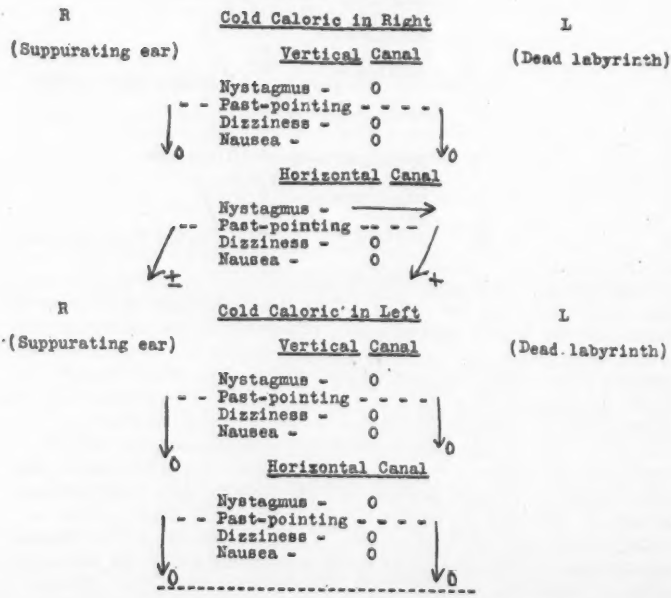
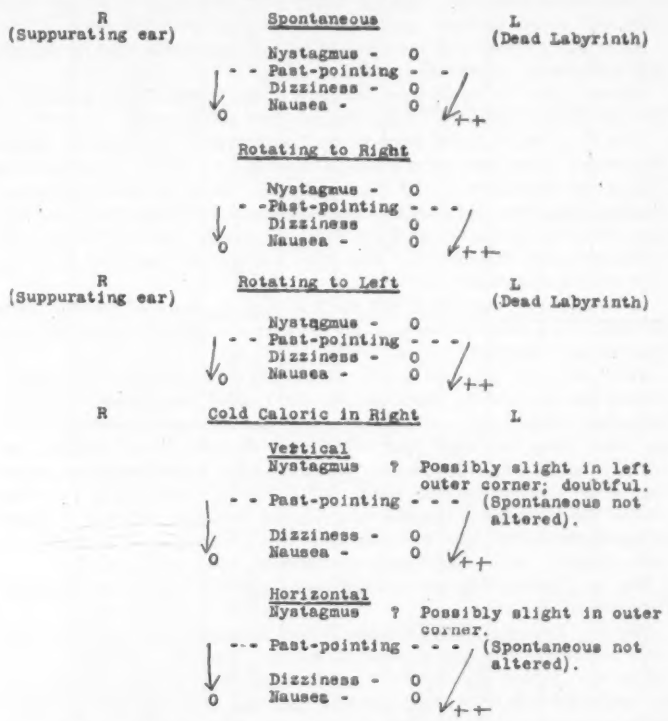


Chart II. J. B.



Subsequent History: May 12, 1920. Right ear has been discharging for some time; during the past two weeks left ear has also been discharging, and man had aching pain back of the ear. Right ear contains large granulations, occluding the canal. Very deaf. Sudden left facial paralysis a few days ago. At times some blurring of vision. O. E.—negative; BS—20/20.

Chart III. J. B.



Shortly after the above observations a simple mastoid operation was performed on the right ear.

June 11, 1921: The right ear had continued to discharge from the wound until three weeks ago, when a large piece of bone came from back of the ear; since then the discharge has ceased. Patient felt well until four days ago; then, sitting down quietly at home, seemingly in usual good condition, he suddenly, on standing up, had severe dizziness, a tendency to fall to the right—which has continued—and nausea at first. The dizziness is not quite so severe now; it came on with a sudden closing of his right ear. The left ear is very deaf following the mastoid on that ear; he has a noise like a faint whistle in the left ear; no pain in ears; dopey feeling in head. Blood pressure, 102-68.

Outstanding Symptoms: Sudden closing of the right ear and at once severe dizziness; hearing reduced in that ear. Blurring vision in walking since present dizziness, which clears when he is not in motion.

August 19, 1921: Has become totally deaf; is very dizzy and complains of pain over left eye. Sight is reducing. A discharge, with odor, from right ear for two weeks. Falls or staggers at once on attempting to rise, from dizziness and in falling *goes to left side.*

Outstanding Symptoms: Lately totally deaf; discharge with odor from right ear; severe dizziness; general weakness; pain over left eye.

Right Ear: Hears with noise box in left ear, but words must be shouted. Slight discharge. Granulation at site of drum.

August, 1921: It would seem that he now has a labyrinth involvement of the second—the right side. Total deafness both sides.

Case V. Case of Acute Suppurative Labyrinthitis Followed by Serous Meningitis Associated with Vertical Nystagmus; Paralysis of Superior Oblique of Homolateral Side; Slight Facial Paralysis and Deafness of Homolateral Side; Homolateral Arm Slightly Incoordinated; Spontaneous Pointing Deviation of Both Arms to Side of Lesion; Fluid Clear—Under Slight Pressure—but Elevated Cell Count; Reduction of Duration of Nystagmus on Turning.

B. R., History: Earache in left ear following an attack of tonsilitis early in April. Two days after earache the ear opened voluntarily and discharged more or less continually for two weeks.

April 16: An aural consultant diagnosed slight mastoid involvement. Paracentesis performed. April 28; the ear stopped discharging. Vomiting and other "gastro-intestinal" symptoms followed. Double vision appeared, one object being seen about four inches lower than the other. Another consultant diagnosed "gastro-intestinal trouble". After bowel movement patient improved. This improvement lasted until following evening, May 1st, when another attack of pain in stomach occurred, and headache; followed by period of improvement until May 4th, when there was a recurrence of headache and "sick stomach", which again gradually subsided.

May 9: Became languid, vomited, and complained of severe headache. Diagnosis of acidosis.

May 10: Patient remained languid; became sleepy, and vomited twice. Another consultant—Dr. Eugene Murray—called. Diagnosis, meningitis.

May 11: Patient admitted to hospital. Complained of constant temporal and occipital headache, with pain in neck; deafness of left ear for past three weeks, with tinnitis and dizziness. (There had been no chill, and temperature was but slightly elevated.)

Examination: "Nystagmus, vertical, especially upon looking to the left. Possible paralysis of external rectus of left eye; left eye lower than right; left pupil larger than right. Total deafness of left ear; left drum normal; no tenderness over mastoid. Slight left-sided facial paralysis." Has lost flesh. Tongue protrudes in median line. No difficulty in swallowing. Incoordination.

Vestibular Manifestations: Vertical nystagmus; spontaneous pointing deviation of both arms to left; left arm co-ordinates imperfectly.

Lumbar Puncture: Spinal fluid clear, under very slight pressure; sugar reduction normal; globulin slightly increased; cell count, 60 per cubic mm. No organisms.

Chart IV, J.B.

	ROTATION			CALORIC				
				Right		Left		
	Spontaneous	To the Right	To the Left	Vertical	Horizontal	Vertical	Horizontal	Date
Nystagmus	0	0	0					Oct. 6, 1921
	0	0	0					Jan. 19,1922
Past-pointing	↘2 in. ↙in.	↘2 in. ↓0	↘2 in. ↓?					Oct. 6, 1921
	↘2 in. ↓0 at times	↘2 in. ↓0	↓0 ↓0					Jan. 19,1922
Dizziness	0	0	0					Oct. 6, 1921
	0	0	0					Jan. 19,1922
Nausea	0	0	0					Oct. 6, 1921
	0	0	0					Jan. 19,1922

Pre-operative Diagnosis: Labyrinthitis, with cerebellar involvement. Abscess?

Operation: Drainage of diseased deep cells of petrous pyramid; opening of semi-circular canals and cysterna of internal auditory meatus. Incision of dura over cerebellum.

Operation Notes: The mastoid was opened and found full of granulation tissue; no pus; the solid angle between sinus and petrous bone opened; small, deep cells found involved and filled with granulation tissue but no pus. Proceeded inward behind posterior semi-circular canals. No bleeding. Was compelled, because of forward-lying sinus, to chisel away posterior arm of posterior semi-circular canal, which exposed both canals well; canals contained no pus and no fluid, pin-point opening found in dura (which may have been operative). Discharge of cerebro-spinal fluid on reaching internal auditory meatus. Dura over this area covered with granulations.

At completion dura was very black; two or three openings in dura from which cerebro-spinal fluid was flowing, but not in great quantity. Middle fossa not exposed; sinus not injured. Middle ear was not opened.

Operative Comment: "It would seem that this child has a serous labyrinthitis—possibly a brain abscess *over* the cerebellar region. It might have been better if the labyrinth had been opened through the middle ear and the cochlea exposed as well as the semi-circular canals. Exploration into cerebellum was not attempted, although the spontaneous deviation—the lack of good co-ordination of the same side—pointed to an involvement of brain stem."

At completion of operation the child apparently could not move its arm on that side, but this difficulty rapidly disappeared. O. E. was negative.

May 12: Temperature, 100-99°, pulse very fast, 118-148, and at times irregular.

May 13: (Second day after operation)—pulse still rapid, 130; temperature, 99-101°. Patient lies on right side; in this position eyes are deviated toward the right and downward; ophthalmoscopic examination negative, but may be slightly indistinctive. Complains of soreness of throat. Slight facial paralysis, about the same as before operation. Left arm moves well but incoordinately; Leucocyte count, 19.200. Polynuclears, 85%; Large Lymphocytes, 9%; Small Lymphocytes, 5%.

May 16: Nurse reports, "She seems to look at you with the right eye." Left pupil larger than the right. O. E. negative, but both discs whitish. Paralysis of left external rectus. Patient has tendency to look downward and to the right; in this position of eyes there is no nystagmus; there likewise is no nystagmus when looking downward and to the left, but rotary nystagmus on looking upward when patient is in recumbent position. Tongue protrudes in median line; palate goes up on right side; patient complains that when she swallows, the solids and liquids "do not go down right". There seems to be a shoulder girdle in which the incoordination is most marked when she has shoulder and elbow free.

Both vocal cords move normally, but there is fluid—mucous—lying in the sinus pyriformis, which causes coughing.

May 18: O. E. negative; temperature, normal; pulse, still fast—118-130.

May 19: Smear and culture from left ear: streptococci and cocci probably from sarcina group.

May 29: Left pupil larger than right; left eye turns in on looking to right; rotary nystagmus to right, but absent on looking to left; when the eye is still (this occurs when either eye is covered). Complete paralysis of the left external rectus. Incomplete facial paralysis. Marked rotary nystagmus in looking upward; left eye slightly lower than right. No dizziness when patient tries to sit up, but in returning to recumbent position she becomes dizzy.

June 7: O. E., negative. The left disc is whiter than the right, but it may be that the right is too red. Incoordination of the left arm. Left complete facial paralysis, cannot close eye; complete paralysis of external rectus; on closing left eye moves right eye inward without nystagmus, on turning it outward to the left there is nystagmus. With both eyes open, on looking upward there is rotary nystagmus of vertical tendency; no nystagmus on moving to left, but of course the left eye does not go outward because of paralysis of left external rectus; left eye is downward, and on moving, raises the upper lid; looking to right no nystagmus. No nystagmus on looking downward, but marked vertical nystagmus on looking upward. Apparently has a paralysis of the superior rectus or of the superior oblique—I think of the superior rectus, but I am not sure. Temperature, normal; Leucocyte count, 9,000. Polynuclears, 67%; Large Lymphocyctes, 7%; Small Lymphocytes, 25%; Eosinophiles, 1%.

June 8: Vestibular Reactions Spontaneous. No spontaneous past-pointing; spontaneous rotary nystagmus to right at short intervals; the nystagmus is more noticeable when the patient looks to the right and somewhat upward. It is more constant in this position than when she looks straight ahead; it is also more marked when she looks upward. When she looks straight ahead the act of blinking seems to increase the nystagmus.

Rotating to Right. (1) Right hand deviated two inches to right—did not deviate more than once. Left hand did not deviate. Dizziness. No nausea. (2) No deviation of either hand. No perceptible nystagmus was induced.

Rotating to Left. No deviation of either hand; horizontal nystagmus to right of 8-10 seconds. Dizziness. No nausea.

Cold Caloric in Left. Head erect; produced no reaction. (Dead labyrinth). Head back; no perceptible nystagmus induced, the possible slightly horizontal to left. Dizziness. No past-pointing.

August 14: Double vision improving. Images 6 inches; at times vision is single. Image separates on looking to the left. The child apparently is perfectly well. Wound healed.

Case VI. Case of Generalized Protective Meningitis, Result of Suppurative Labyrinthitis, with Vestibular Manifestations of Increased Intracranial Pressure—Non-Reactibility of Opposite Vertical Canal to Cold Caloric—from Which a Diagnosis of Increased Intracranial Pressure was Made; Reduction of Duration of Nystagmus on Turning; Inability to Cause Arms to Deviate Toward Side of Lesion; Lumbar Puncture Revealed Pressure and High Cell Count. Following Operation Severe Symptoms of Meningitis.

J. G., Male; adult. Seen with Dr. Baldwin November 24, 1919.

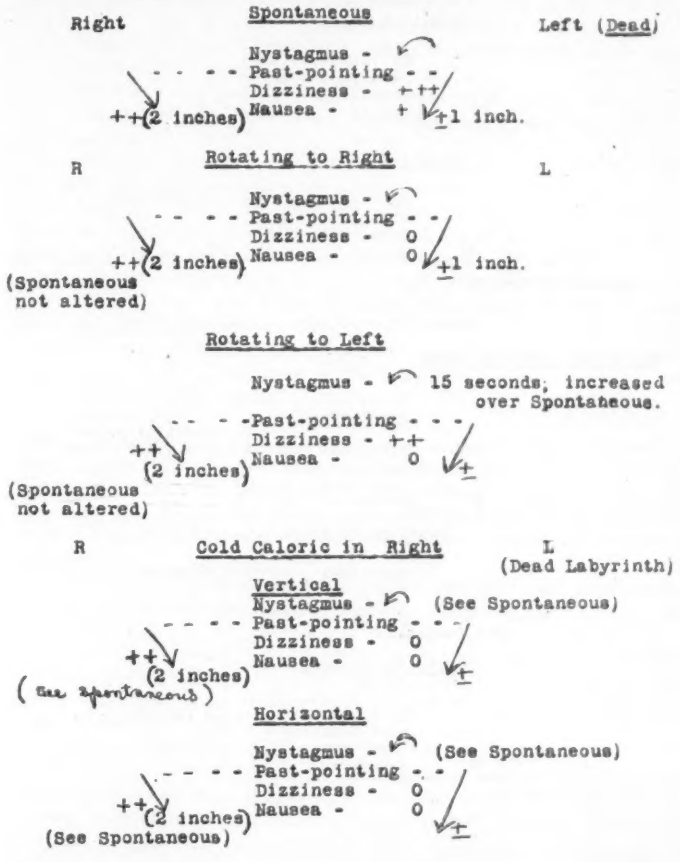
History: Had discharging right ear for three months. For the last three weeks had pain in right ear with severe pain in head in right temporal regon; attacks of dizziness during past three weeks. "I shall be going and

suddenly something occurs and I sway. I have never fallen but have run into things." Does not know to which side he sways. Several chilly sensations and two attacks of vomiting. Last two days facial paraysis.

Examination: Right drum membrane destroyed; granulations and pus in external canal. No hearing in right ear.

Chart I. G. C.

Graphic Representation of Above April 30:



Rotating to Right: Horizontal nystagmus to left of five to nine seconds. No dizziness or nausea. Wide deviation of right to right (10 inches). Slight deviation of left to right (3 inches).

Chart II. G. C.

Graphic Representation of the Above:

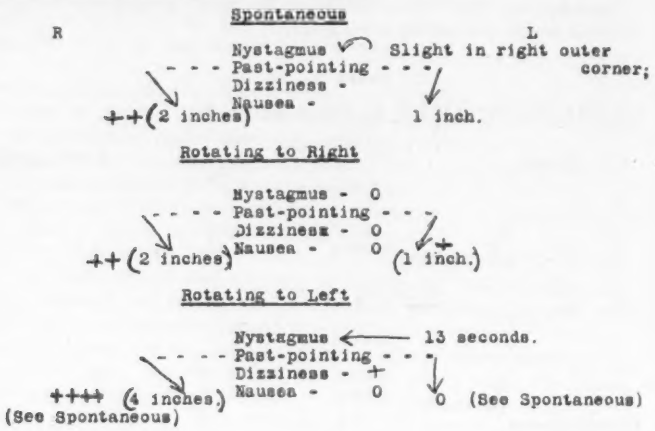
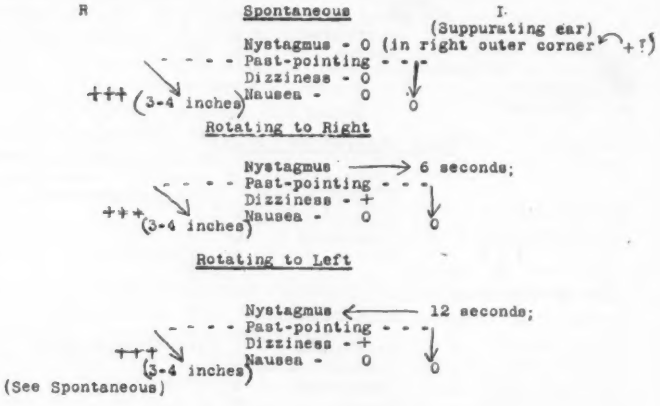


Chart III. G. C.

Wednesday, June 29, 1922:



Hearing:

	Right ear:	Left ear:
Watch -	Six feet	Totally deaf.
Whisper -	Across room	
Voice -	Across room.	

Rotating to Left: Horizontal nystagmus to right of five seconds. No dizziness or nausea. No deviation of either hand; quick movement and large amplitude; wide pointing deviation of both hands to left; dizziness.

Falling.

Rotating to Right: Head forward; on straightening up, fell to right; dizziness and nausea.

Rotating to Left: Head forward. Did not fall; very slight dizziness.

Cold Caloric in Right: Dead labyrinth.

Cold Caloric in Left: Produced no nystagmus—if any it was horizontal to left—no pointing deviation of either hand, no dizziness or nausea.

Throwing Head Back: Produced a violent horizontal nystagmus to right.

Temperature, 99°. Pulse, 76.

November 27: First lumbar puncture performed; fluid clear, under pressure. Globulin increased. Fehling's slightly reduced. Cell count, 100 cm. No organisms.

November 28: Second lumbar puncture; fluid clear, under pressure; globulin increased; sugar reduction not normal; no organisms.

November 29: Operation. Labyrinth opened; fistula found; granulations. Neumann's operation. Posterior fossa opened behind posterior pyramid; a quantity of cloudy cerebro-spinal fluid evacuated.

Following operation patient had severe pain over left eye, numbness of fingers of left hand; was restless at night; temperature 101°-103°; Leucocyte count, 12,800; Polynclears, 73%.

December 3: Stiff neck; severe pain in head; Leucocyte count, 16,400; Polynuclears, 85%; smear from wound, staphylococcus, streptococcus mucosis, capsulatic Gram—bacilli.

Temperature, 102°-104°; pulse slow, 82-90.

From December 3 to December 28 patient continued with restlessness at night, slightly delirious with alternating attacks of stupor, from which he could easily be aroused; severe pain in head; stiffness of neck; Kernig; pain behind left eye; photophobia with a continuously high temperature, 101°-105° —and a proportionately slow pulse. Several blood cultures were negative. Leucocytosis.

December 26: Third lumbar puncture; no pressure; color pale yellow; globulin greatly increased; sugar reduction, about one-half normal; cell count, 633 cm.

December 31: Patient seemed better in every way. Temperature 99-101°.

January 7, 1920: Patient looked very ill; pulse very fast and of poor quality, but rested better at night; still complained of stiffness in back and legs, but head felt better.

January 18: Patient much better in every way; temperature normal.

February 24: Patient discharged from hospital feeling fairly well but with a discharging ear. A few days later he discharged a sequestra of cochlea, after which the ear promptly healed and all symptoms disappeared.

A similar case with dead labyrinth, high temperature, delirium, almost maniacal headache, double papilloedema, cloudy spinal fluid, with cell count of over 3000 but without micro-organisms, an extenteration of labyrinth, with incision of the dura, was promptly followed by recovery.

Case VII. Result of Cleaning out Deep Cells of Petrous Developed Labyrinthitis and Serous Meninigitis with Cell Count of Lumbar

PUNCTURE FLUID SPONTANEOUS ROTARY NYSTAGMUS AND INWARD POINTING DEVIATIONS OF BOTH ARMS, THE DIRECTION OF WHICH COULD NOT BE ALTERED.

(1) Absence of reaction to cold caloric of opposite vertical canal (from increased intracranial pressure) and (2) reduction of duration of induced nystagmus to fifteen seconds giving rise to the supposition that the functional test ultimately would return to normal.

History: Boy, G. C. Seventeen years of age.

February 22: Scarlet fever, and following this, about February 28, had earache on left side and discharge, when patient had delirium, and at least one chill. During discharge, had headache over temple. Followed by period of improvement but had discharging ear.

April 12: Mastoid operation by Dr. Leifeld, sinus and dura exposed; apparently improved; no temperature, no headache, until April 21, when patient had slight rigor (possible from ice cream). Next day *pain behind left eye,* which continued on and off. Discharge from ear profuse; no dizziness; temperature, 100.2°-102°; nauseated and vomiting.

April 26: Lumbar puncture; fluid clear, not under pressure; globulin sligtly increased; cell count 5; no organisms; blood culture, negative; O. E. veins full, no indication of injection of disc. Hearing, greatly reduced but present in left ear.

April 27: Second Operation. Eradication of deep cells of petrous. Decided not to open his dura. At completion of the operation I was not entirely satisfied; "the boy probably has a serous meningitis, although the low cell count—only three—would signify otherwise."

Granulation tissue in the mastoid wound. The deep mastoid cells behind the external semi-circular canals also opened. There was a large amount of carious bone along the zygomatic process where the dura was freely exposed. The sinus was covered by a thick granulation. Made a small opening in the sinus; had bleeding, but not very free.

April 28: Vomited. Had some dizziness following the operation. "He does not look entirely right." He had a white cell count of 17,000. Blood culture, negative. "I am still of the opinion that he has an involvement of the temporo-sphenoidal lobe."

April 29: "Since the operation the boy has been slightly dizzy, and on changing his position the dizziness is increased." Has a nystagmus on looking toward the right (away from the lesion). "It is possible that during the operation there was some injury done to his labyrinth, although I think not." Today he appears slightly dopey, but his mother says he always sleeps considerably when sick. Temperature, 101°. "I am afraid he is developing serous meningitis. It may be this is due to the injury of his labyrinth, because the deep cells of the apex of the petrous portion of the temple undoubtedly were involved."

Second Lumbar Puncture: Fluid under great pressure; macroscopically clear; globulin, slightly increased. Sugar, normal. Cell count, 322.5 per cubic mm. Smear shows no organisms.

April 30: Temperature, irregular—90.2—102°. Appears much better; is not dizzy. Total deafness of left ear.

Vestibular Reactions Spontaneous: Rotary nystagmus to right on looking straight ahead; more noticeable on looking in right outer corner. Spontan-

eous deviation of *right hand two inches to left.* Tendency for left hand to deviate to *right.*

Rotating to Right: No perceptible change in the spontaneous rotary nystagmus; (possibly stopped for three or four seconds). No dizziness or nausea. Right hand deviated to left (see Spontaneous Deviation). Left hand deviated to right (see Spontaneous Deviation).

Rotating to Left: Spontaneous rotary nystagmus markedly increased for about 15 seconds; then about the same amplitude as before rotation. Dizziness. No nausea. Right hand deviated to left (no increase over spontaneous deviation): Left hand deviated to right (see Spontaneous Deviation).

Cold Caloric in Right:

Head erect: The spontaneous nystagmus to right was not altered; no change in spontaneous deviation; right deviated to left—left deviated to right; no dizziness; no nausea.

Head back: No perceptible change in spontaneous nystagmus; right hand did not deviate (see Spontaneous Deviation); left hand deviated to right.

Culture, sterile after 24 hours. Differential, Polynuclears, 93%. Lymphocytes, 7%.

May 2: Patient much better in every way, but still has irregular temperature—102-102.2°. No dizziness. Leucocyte count, 13,400. Differnetial, Polynuclears, 78%; L. Lymphocytes 10%, S. Lymphocytes, 11%; Eosinophile, 1%. Blood culture taken; negative. Spinal fluid; slight opacity of fluid, considerable pressure; globulin slightly increased. Sugar, normal. Cell count, 842 per cubic mm. No organisms in smear. Culture. Differential, Polynuclears, 95%; Lymphocytes, 5%.

May 5: Spinal fluid under considerable pressure; fluid, opalescent and showed slight flocculent precipitate. Globulin, slightly increased. Sugar, normal. Cell count, 600 per cubic mm. No organisms in smear. Fluid cultured; no organisms. Differential, Polynuclears, 90%; Lymphocytes, 10%.

May 6: Leucocyte count, 8,800. Differential, Polynuclears, 72%; L. Lymphocytes, 13%; S. Lymyphocytes, 14%; Eosinophiles, 1%. "This is the third lumbar puncture which has been done. At the first one before the operation for his labyrinth he had no high cell count. A short time afterward he had a cell count of 150. Three days later it rose to over 300. It is now over 600. The boy, however, looks perfectly well. He is pale. He has no other symptoms. In view of the fact that none of his labyrinthine symptoms came on until after the operation, and we know that he heard before that, that his nystagmus developed the following day, and that he is dizzy, we had decided that it must be a protective process. We are impressed with this idea because of our experience with case J. G. (Case IV), who after a labyrinthine operation, continued to have a high cell count with some rigidity of the neck. However, one month after the operation, he still had a cell count of 600. Some weeks later a small piece of sequestra from the labyrinth came away, and the man's condition immediately cleared up. This makes us think that in a chronic case there is a protective meningitis going on along the posterior surface of the petrous portion of the temple. In opposition to this, however, is the fact that he had before the operation a severe pain behind his eye, that he had one chill and was delirious.

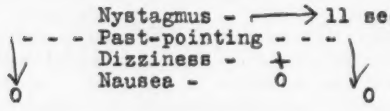
"The diagnosis of the case must remain between three things; one, of protective meningitis, the result of the labyrinthine operation and the de-

Jan. 11, 1921: Chart I. M.A.

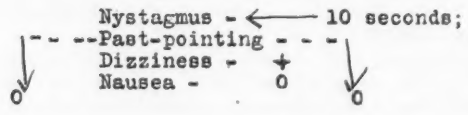
Vestibular Reactions;

No spontaneous nystagmus or past-pointing.

R. Rotating to Right: L



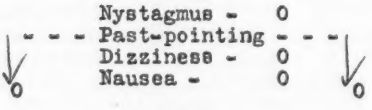
Rotating to Left



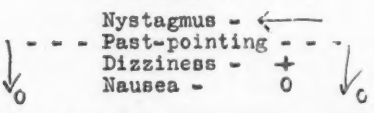
Cold Caloric in Left

R L

Vertical



Horizontal



velopment of a protective area so as to cut off the ductus endo-lymphaticus, or the internal auditory meatus tract; second, an encapsulated abscess of the temporo-sphenoidal lobe, now at least of from four to six weeks' duration; or third, an encapsulated serous meningitis, either along the posterior portion of the petrous or over the tegmen."

Vestibular Reactions:

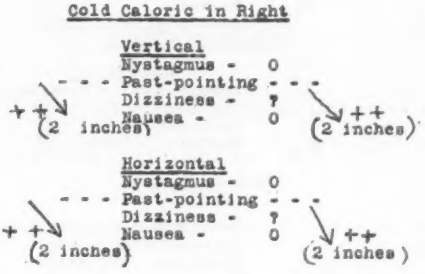
Head erect: Spontaneous, slight nystagmus with rotary tendency to right in right outer corner. Spontaneous deviation of right 2 inches to left. Spontaneous deviation of left 1 inch to right.

Rotating to Right: No perceptible nystagmus; no dizziness or nausea. Left hand deviated to right; right hand deviated to left. (See Spontaneous Deviation).

Rotating to Left: Has nystagmus to right, of good amplitude, lasting 13 second. Slight dizziness; no nausea. Right hand deviated 4 inches to left. Left hand did not deviate. (See Spontaneous Deviation).

Chart II. M. A.

January 13:



May 7, 1921: Totally deaf. Slight nystagmus in looking to right; no adiokokinesis; no hemianopsia of color on rough test; no pointing deviation; O. E., about the same; veins, full but not tortuous.

Hearing:

Right ear: Watch, six feet; whisper, across room; voice, across room. Left ear: Totally deaf.

It would seem from studying the foregoing four cases that immediately on the increase of the intracranial pressure in protective meningitis or intrapiarachnoid abscess of the anterior surface of the cerebellum, (1) the nystagmus normally induced from the remaining functioning end-organ is reduced from the normal sixteen seconds to about half that duration—namely, to eight or nine seconds—and (2) the spontaneous past-pointing of the arm caused by the involvement of the cerebellar cortex situated posterior to the bony labyrinth cannot be altered by stimulation. Both of these observations would suggest a localizing diagnosis of the lesion.

A study of these cases furnished me a clue to that larger class of cases now so frequently seen, which—following an injury, such as fracture of the skull with cerebral trauma, or associated with a chronic progressive deafness, or from intoxications of all kinds, syphilis, and so on; in fact, any condition which lessens the reactibility of the horizontal fibres to particular forms of stimuli (repeated turnings or repeated aerial flights, the clinical signs of which rapidly disappear)—immediately develop a great reduction in the duration

Chart III. M. A.

Feb. 18:

R L

Spontaneous

Nystagmus ⟶ Slight, in left outer corner.
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus ⟶ Possibly 7 secs;;slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Left

Nystagmus ⟵ 7 or 8 secs.; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Cold Caloric in Right

R L

Vertical
Nystagmus -
Past-pointing - - -
Dizziness - ++
Nausea - 0

Horizontal
Nystagmus - ⟶
Past-pointing - - -
Dizziness - ++
Nausea - 0

Cold Caloric in Left

Vertical
Nystagmus - ↖
Past-pointing - - -
Dizziness - +
Nausea - 0

Horizontal
Nystagmus - ⟵
Past-pointing - - -
Dizziness - +++
Nausea - 0

February 24:

Wasserman - negative.

Chart IV M.A.

Feb. 29: Vestibular Reactions

Spontaneous

R L

Nystagmus ⟶ Slight in left outer corner;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus ⟶ Possibly 7 seconds; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Left

Nystagmus ⟵ 7 or 8 seconds; slow movement;
Past- pointing - - -
Dizziness - 0
Nausea - 0

Cold Caloric in One Ear - the Right

Vertical

Nystagmus - ↷
Past-pointing - - -
Dizziness - ++
Nausea - 0

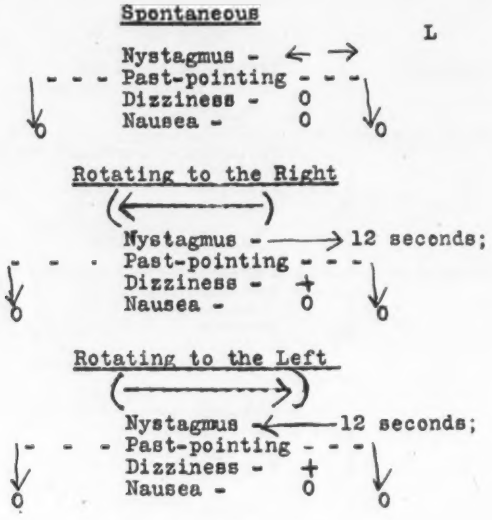
Horizontal

Nystagmus ⟶
Past-pointing - - -
Dizziness - ++
Nausea - 0

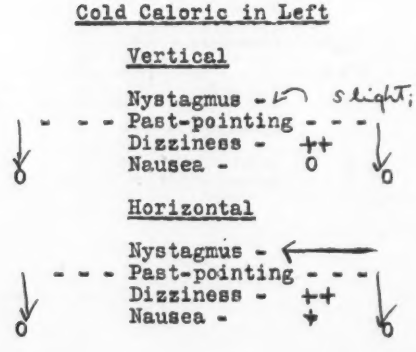
Vestibular Reactions.

Chart I. J.M.

December 31:



January 27:



of the nystagmus on turning; showing that while the fibres of the vertical canals apparently are much earlier attacked by an increase of the intracranial pressure when stimulated by the cold caloric, the horizontal fibres also are easily affected by any disturbance, as far at least as stimulation by turning is concerned.

Pointing Deviation:

B (Case IV) had bilateral past-pointing away from the lesion; homolateral—pointed inward; contralateral—pointed outward, with falling away from the lesion.

The spontaneous pointing deviation could not be altered by the entire turning or caloric. After the opposite labyrinth became affected the homolateral arm of the second labyrinth was altered and pointed inward (away from the second lesion toward the first lesion), and again this spontaneous deviation could not be altered. In B the contralateral side apparently lost the capacity for stimulation. It stopped deviating in May, 1920; the contralateral continued reactible for a time.

C (Case VII) had bilateral past-pointing deviation *inward*, which could not be altered. The contralateral side had a greater pointing deviation than the homolateral side. The homolateral side later could not be made to deviate at all, while the contralateral side still continued inward.

R (Case V) there was no past-pointing deviation, but the records are so few, and the child was so sick that it cannot be accepted as of any great value.

These four cases in a general way confirm the opinion that the spontaneous past-pointing deviation cannot be altered until at least the lesion has been removed as in the cases reported by Lowenstein. He, with Bárány, thinks there is a spontaneous past-pointing deviation of the homolateral side outward.

The fact that B, C and R could not have the spontaneous past-pointing deviations altered long after the inflammatory process had subsided shows that in these cases of protective meningitis the cerebellar cortex is affected (by the inflammatory process).

These cases would certainly tend to show that whatever mechanism is involved by turning, in diffuse suppurative disease in the meshes of the piarachnoid over the cerebellar cortex, this mechanism is suddenly impaired to such an extent that it does not recover. The influence of this past-pointing deviation would appear the same for both the spontaneous homolateral and contralateral—the past-pointing deviation could not be altered; and in some of the cases at least

the homolateral deviation continued reactible while the contralateral was non-reactive.

II. *The Diagnostic Value of a Reduction of Intracranial Origin in the Duration of the Nystagmus from Turning in Diffuse Lesions of the Posterior Fossa:*

I am persuaded that in the cerebellar cortex of the posterior fossa there is something which has to do with this. It is probably in the cerebellar cortex because cortical lesions are the ones that induce it quickly (as in the cases reported), while in intra-cebellar lesions such as intra-cerebellar abscess, this duration of the obtainable nystagmus from turning is not affected.

My own experience with this reduction in cases of fracture of the skull of the posterior fossa, leads me to believe that it is the filling of the piarachnoid pathways of the posterior fossa with either exudate (as in the herein reported cases of protective meningitis) or with blood as in linear fractures of the skull, chiefly of the posterior fossa; that gives rise to inflammatory and reactive changes which affect the cerebellar cortex; for the reduction of the duration of the obtainable nystagmus by turning is immediate, as occurred in a case of meningitis which recovered after subarachnoid irrigation. The reduction in the deviation of the nystagmus was demonstrated one week after the onset of the disease. When, however, the exudate is chiefly confined to the cerebrum, the reduction of the induced nystagmus from turning either does not occur at all or is permanent, as another case of suppurative meningitis which recovered following cerebral and spinal subarachnoid irrigation, had a normal deviation of the nystagmus a few months later.

Induced Nystagmus from Turning and from Heat or Cold Physiologically and Diagnostically Different:

From a misconception we have in the past regarded the induced nystagmus from turning and from caloric as identical. This is physiologically and clinically incorrect, as I have herein demonstrated. There is no evidence to substantiate the view that the induced nystagmus from turning and from cold and heat are identical. We have presumed, however, that they were identical, because objectively they are apparently the same; but judging from physiological analogy it is much more likely that they are in certain parts, at least, separate. The forms of stimuli are so dissimilar—turning being a mechanical stimulus which is well adapted to cause a molecular disturbance in a fluid medium (such as the fluid of the labyrinth) analogous to a certain degree to stimulation of the proprioceptive

sensory system—deep sensation—while cold and heat are thermic stimuli ill adapted to such disturbance, but, on the other hand, well adapted to stimulation of the nerve fibres by cutaneous sensation. As both are forms of sensory stimuli, the vestibular apparatus being one of the early specialized apparati to convert sensory impulse into voluntary and involuntary muscular contractions, the different sensory impulses from turning and caloric probably are transmitted by the vestibular apparatus in a similar way as the transmission of cutaneous impulses to the cord from pressure or thermal sensations, either by a different set of cutaneous nerves, or at least by nerves differentiated in the posterior roots.

It is possible that the cerebellar cortex *as a whole* has an analogous function to the posterior roots as far as the differentiation of the vestibular stimulation or impulses is concerned. In turning we are stimulating in a way a muscular sense—the sense of movement; while in caloric we are stimulating a sensory sense—a cutaneous (thermic) sense.

TWO CASES ILLUSTRATIVE OF THE IMMEDIATE REDUCTION OF THE INDUCIBLE NYSTAGMUS FROM STIMULATING THE HORIZONTAL SEMI-CIRCULAR CANALS BY TURNING WITH PRESERVATION OF NORMAL HEARING IN DIFFUSE TRAUMATIC LESIONS OF THE POSTERIOR FOSSA, SUCH AS FROM LINEAR FRACTURE OF THE SKULL.

Case VIII. M. A., a girl, 8 years of age.

Resumé of Case:

Struck by an automobile; since then has had dizziness and headache, double vision in the distant, not in the near, which may not be pathological but looks abnormal; high degree of hypermetropia with astigmatism; reduced vestibular reaction in the horizontal canal; absence of reaction in vertical canal in left ear; hearing normal.

Impression:

It would seem then that she undoubtedly had a fracture somewhere through into the posterior fossa. Both vertical and horizontal canals fail to function properly.

History:

In March, 1920, was run down by an automobile and badly injured, especially on the back of her head. She was taken to a drug store and about an hour afterward taken home in an automobile by a policeman. The mother does not know whether or not the child was uncoscious at any time. The physician who was called in to attend her and helped her home, attended her for a few weeks at her home. The back of the child's head was much swollen and discolored for more than a month, but the doctor did not regard it as due to a fracture of the skull. The child was in bed for a week after the accident. On getting up she had to be led, but the mother was unable to say whether this was due to dizziness, to general weakness, or to the injury to her right leg.

Patient seemed to recover gradually, but remained very pale, had no appetite, complained of pain beginning at the back of her head and coming

over to the eyes, and said she was invariably dizzy on first standing up. Since the accident she has been told at school that her sight is not normal. She does not see writing on the blackboard well, and her head aches after reading for a time. When asked if she ever sees double, she replied that "when things are far off they are two." A street car at a distance appears to be two cars, but becomes one as it draws near. The same is true of an automobile; also of persons; approaching at a distance she sees two, but when they are near by she sees them normally.

When she becomes excited or worried she feels a soreness or stiffness in her neck and an aching in her left ear. She did not seem to have any ear symptoms at the time of the accident.

January 13, 1921:

Hearing:

Watch: right ear, 6 feet; left ear, 6 feet. Whisper, right ear, across room; left ear, across room. Voice: right ear, across room; left ear, across room.

Tuning Fork:

Normal duration of fork: A. C., 210 seconds; B. C., 140 seconds.

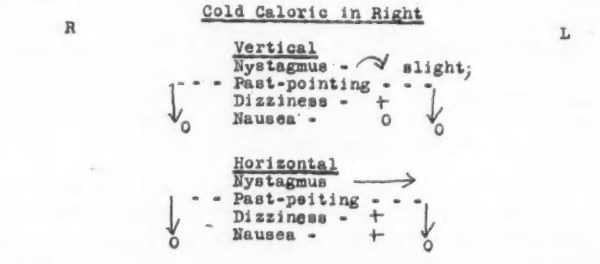
Right ear, A. C., 200 seconds; B. C., 140 seconds. Left ear: A. C., 200 seconds; B. C., 140 seconds.

Weber: Vertex: Sound referred alike to each ear. Frontal: Sound referred to left ear. Teeth: Sound referred to left ear.

Galle: Sound referred to closed ear in all cases.

Chart II. J.M.

February 10:



February 16:

Report of Roentgen Examination:

"Plates were made of the head in both lateral positions and antero-posterior position with the occiput on the plate.

"They show a small line of fracture on the right side in the occipital bone just below the occipito-parietal suture. This is seen in both right lateral and antero-posterior views."

Case IX. J. N., fireman, 26 years of age.

History: Previous history of good health, with no history of acute illness. Fell from third story window when infant of three years. Nov. 24, while driving engine which collided with trolley car, patient was thrown from engine to street. Was admitted to hospital suffering from abrasions, contusions and possible fracture of the skull.

Examination: General appearance of well-nourished, well-developed white male of 26, not acutely ill, lying quietly in bed, conscious and rational.

Head: Scalp, skull and hair, negative.

Eyes: Contusions over left eye, with a sub-conjunctival hemorrhage; pupils equal, regular, react to light, sclera clear in right eye, no nystagmus, no strabismus. Eye-grounds negative. No facial paralysis.

Neck, heart, lungs, abdomenn, extremities, skin, all negative; nodes, not palpable.

Reflexes: Knee and ankle, active and equal; no Babinski; no clonus; no Oppenheim; abdominals and cremasteric active and equal. B. P. 140 over 90.

X-ray showed fracture right occipital region; no depression.

Provisional Diagnosis: (1) Contusion of left eye; (2) possible fracture of skull.

Remained in hospital from November 24 to December 3, when he was discharged.

After leaving hospital he complained of shooting pains from the top of his head to his neck, the pains lasting but a few minutes. Examination revealed dilated veins of the conjunctiva of the right eye, which were not noticed before. O. E. was negative.

Note on Vestibular Reactions: Cold caloric in left ear gave nystagmus but no pointing deviation. It seemed impossible to cause a pointing deviation in either arm. Both of the vestibular reactions were very much reduced.

The fact that the reduction of the turning is of central, not perpheral, origin, was shown by the observation of Young*, who found that in cases of heritary deaf-mutism the duration of the nystagmus in turning was not reduced, while in cases of deaf-mutism from a central disease, such as follows cerebro-spinal meningitis, it was not possible to cause an induced nystagmus on turning. Syphilitic deaf mutes may have a normal duration of the nystagmus, or no nystagmus at all, depending apparently upon the site of the lesion, whether peripheral or central.

That the reduction of the duration of the obtainable nystagmus may be due to intracranial involvement as well as readjusted compensation, and that it may occur immediately upon invasion of the meshes of the subarachnoid spaces by exudate or inflammation are shown by a case of protective or tubercular meningitis referred to previously, treated by subarachnoid irrigation, rotated on the fifth day after the cerebro-occipito-allantoid irrigation and within a week after the appearance of the meningeal symptoms. In this case, with

Griffith (Laryngoscope. January, 1920, p. 22. and March, p. 129) demonstrated experimentally what had been clinically proven by Dunlap (Jour. A. M. A., Vol. 73, No. 1, p. 54) that repeated turning reduced and finally abolished the induced nystagmus as well as the vertigo and past-pointing.

Robertson, C. M. (Laryngoscope 32-105, Feb. 22. reviewed in Int. Educ. & S. Jour., April. 1922, 8b-157) also found reduction of nystagmus on repeated flights and turnings.

*Young, Calvin: Vestibular Reaction in Deaf-mutism; Journal Laryngology and Otology, 1921, Vol. 36, No. 11, p. 524.

normal hearing the induced nystagmus obtainable was reduced to 8 and 13 seconds.

It is possible that the reduction occurs when the virulence of the infection is confined chiefly to the meshes of the subarachnoid spaces of the posterior fossa in the cases herein reported; while in the case of P*, the inflammation following the line of fracture was confined mainly to the subarachnoid spaces of the cerebrum. In this instance, the pus and bacteria found in the lumbar puncture may not have been uniformly diffused over the cerebellar cortex to the extent and degree as when associated with protective and hemorrhagic lesions of the posterior fossa itself.

Conclusion and Deductions:

(1) In cases of increased intracranial pressure there is a reduction of the reactibility of the vertical canals of the side opposite the lesion, as evinced by the cold caloric when applied in the upright position with the head tilted forward 30°;

(2) When the lesion is situated above the tentorium, however, the reactibility, although reduced or delayed, is generally attainable. In increased intracranial pressure of the *posterior fossa* the reduction amounts to a complete absence of reactibility of the vertical canal of the unaffected side;

(3) This vestibular manifestation of increased intracranial pressure—namely, reduction of and in the posterior fossa, absence of reactibility of the vertical canals of both ears to the cold caloric—is a general diagnostic symptom of importance but is of no localizing value. Its presence, on the other hand, largely prevents further localization by the vestibular tests alone;

(4) A protective collection of abnormal cerebro-spinal fluid with partial obliteration of the cerebro-spinal fluid circulatory system pathways from labyrinthine involvement, either suppurative or traumatic, situated over that portion of the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid and manifested by a protective meningitis—shown by a high cell count but without micro-orgnaisms in the lumbar puncture—has a definite vestibular symptom complex.

(5) This vestibular symptom complex is:

(a) Loss of reactibility of the vertical canal of the opposite side, as well as of the affected side, from increased intracranial pressure;

(b) Early reduction of the maximum duration of the induced nystagmus, from stimulation of the opposite side from turning;

Eagleton, W. P.—Subarachnoid Irrigation for Suppurative Meningitis—Trans. Amer. Otol. Soc., 1921.

(c) The duration of the nystagmus from turning being only about 50% of that obtainable after destruction of one labyrinth; the reduction, therefore, is of intracranial and not labyrinthine origin;

(d) Both vestibular manifestations appear sufficiently early to be of diagnostic value and continue for a considerable period, and in certain cases are permanent;

(e) The spontaneous past-pointing deviation from the intracranial involvement, rapidly disappears, probably from the establishment of compensation;

(f) Clinical evidence of the earlier presence of a spontaneous past-pointing deviation can be elicited by turning; as after a considerable period of time—after several years in one of my cases—it was impossible to induce a deviation in the opposite direction;

(g) The permanence of the latter vestibular manifestation—inability to induce past-pointing in the opposite direction to a previous spontaneous deviation—may explain the unreliability of past-pointing deviations in general, if further observation confirms the suggestion here offered that inflammatory and infiltrating lesions of the central portion of the vestibular apparatus primarily accompanied by spontaneous past-pointing deviations—possibly are never wholly recovered from and are later manifested by an inability to produce an induced past-pointing deviation in the opposite direction; while spontaneous past-pointing deviations from pressure such as that caused by tumors, endothelioma, cycts, and extradural hemorrhages, may entirely disappear.

(h) In the presence of a suppurative or traumatic lesion of one labyrinth, absence of reactibility of the opposite vertical canal to cold caloric, as well as of the affected side (associated with a 50% reduction of the maximum attainable duration of the inducible nystagmus from stimulation of the unaffected horizontal canal by turning—with or without an associated spontaneous pointing deviation), is a symptom complex of intracranial involvement of diagnostic value and suggests a protective meningitis with localized serous effusion—situated on the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid near the internal auditory meatus.

(6) As intrapiarachnoid abscess from labyrinthine suppuration frequently is located in this situation, it is suggested that possibly the same vestibular symptom complex may be present here also; in this case—although this suggestion is purely from analogy and as yet has no clinical evidence to support it—this vestibular symptom

complex would be of localizing value in distinguishing intrapiarachnoid abscess from intracerebellar abscess.

(7) The induced nystagmus from turning and caloric are fundamentally different because of the difference in the nature of the stimulation;

(8) Consequently, in diseases of the nervous system they should be regarded independently of each other, as they are capable of furnishing separate localizing diagnostic information.

(9) In the cerebellar cortex, or in that portion of it which is covered or reached by the meshes of the piarachnoid, is located a part of the mechanism which has to do with the *duration* of the induced nystagmus by turning. Choking of these meshes by inflammatory exudate (as in protective meningitis from labyrinthitis or from hemorrhage complicating linear fracture of the skull) reduces the duration of the inducible nystagmus immediately by about 50% of the normally obtainable nystagmus from turning. This sign is of great general diagnostic and medico-legal value.

(10) This reduction of the duration of the nystagmus from turning may be a fatigue (non-sustained), a muscular phenomenon, which is now recognized to be a part of the cerebellar functions of asthenia and astasia.

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PREVALENCE OF HAY-FEVER.

Dr. Wm. Scheppegrell, New Orleans, La.

While it is generally admitted that hay-fever is a relatively common disease, there is widespread uncertainty as to its actual frequency. The reasons for this are that the percentage of cases vary greatly in different localities, and that it is only within the last ten years that hay-fever has been recognized generally as an entity.

An illustration of the lack of recognition of typical hay-fever is shown by the following record of the American Hay-fever Prevention Association. When we made an application to the California State Board of Health for co-operation in the control of hay-fever weeds, the president replied that, while hay-fever was common in California and he was desirous of co-operating, the common ragweed, which was the special object of our efforts of control, was not found in California. As soon as the incriminating pollen could be determined, the board would willingly assist in our efforts.

When this report was received, the representatives of our botanical department was instructed to forward to our laboratory specimens of all the most common wind-borne pollens. This soon resulted in demonstrating that the wormwoods (*Artemisias*) replaced the common ragweeds of the eastern states in being the most common cause of hay-fever.

When we announced this discovery, it was contested on the grounds that while the wormwood (*Artemisia tirdentata*) covered vast areas on the western slope of the mountains in Colorado, there was no hay-fever in that section. A representative of the Association was thereupon sent to make an investigation, who soon found that many people suffered from what is popularly called "mountain fever." This has the typical symptom of hay-fever and is prevalent from August to November, the period of pollination of the wormwood. Subsequent investigation confirmed the decision that the so-called "mountain fever" was hay-fever, due to the pollen of the *Artemisia tridentata*.

The records of the American Hay-fever Association, based on extensive reports and investigation, indicate that the number of hay-fever cases in the United States is about 1,050,000, or about one per cent of the total population.

As practically all cases of hay-fever (95%) occur between the ages of 10 and 60 years, and the United States census of 1920 shows that 70 per cent of the population are between these ages, this indicates that 1½ per cent of persons between 10 and 60 years are victims of hay-fever.

In June, 1916, at the request of the American Hay-fever Prevention Association, a questionnaire, under the supervision of the United States Public Health Service[1], was mailed to all the physicians of Louisana, the analysis of which indicated that the number of hay-fever subjects in Louisana is about 1 per cent of its population. Our reports from other sections also indicate that this is a fair average for the eastern states generally.

West of Kansas, hay-fever was formerly supposed to be uncommon, but our investigations have shown that hay-fever in this section, while not as common as a whole as in the eastern and southern states, is by no means infrequent, and is, moreover, constantly increasing. Reports from California and Colorado show that hay-fever is a common disease in these states[2]. Our correspondent in Santa Fe, New Fexico, states that there is a "great deal" of hay-fever in that section. Another from the state of Washington reports that hay-fever is exceedingly common. Our records also show that hay-fever is common in Montana, Oregon, Idaho, Arizona, Wyoming, Utah and Nevada[3].

Hay-fever is far more prevalent in the United States than in Europe. In the former the autumnal hay-fever is much more common, while in Europe the spring form is the prevailing type.

While the average percentage is fairly constant in the large cities generally, there are certain localities in which by reason of the great prevalence of hay-fever weeds or of the great reactional activity of their pollen, the prevalence is as high as ten per cent. In areas when there is a large population of the marsh elder, *Iva ciliata*, for instance, the proportion is especially high. This applies to certain localities in the northeastern section of Louisiana, and to some extent in moist soil from Illinois to Nebraska and south to Louisiana and New Mexico.

HAY-FEVER WEEDS IN CITIES AND TOWNS.

One of the most important causes of hay-fever in the United States is the large area of unused land in cities and towns. These vacant lots are held in the expectation of building on them, or more frequently selling them at some future time at a profit. In the meanwhile, the owner is indifferent as to their appearance or effects, and allows them to become covered with all kinds of weeds.

The seed of these weeds are sometimes the heritage of former cultivation, but more frequently are introduced by cleanings from stables, from garbage, sweeping and refuse, containing the seeds. Most of these weeds are migratory, many of these seeds being distributed by winds or by burs. The ragweeds, cockleburs and pig-

1 2

1. Important hay-fever weed. (These illustrations are from the author's work on "Hay-fever and Asthma," Lea & Febiger, 1922. Common ragweed, Ambrosia elatior, the type of hay-fever weeds.

2. Important hay-fever weed. Giant ragweed, Ambrosia trifida, a common cause of autumnal hay-fever in moist sections.

3 4

3. Important hay-fever weed. Pasture sagebrush, Artemisia frigida, a cause of hay-fever from Minnesota to Saskatchewan, Idaho, Nebraska, Texas and Arizona.

4. Important hay-fever weed. Great ragweed, Iva xanthifolia, a cause of hay-fever from Ontario to Assinoboia, south to Wisconsin, Nebraska, New Mexico and Utah.

weeds, in addition to the wild grasses, are the most common hay-fever plants found on vacant lots east of Kansas.

In addition to the injurious effects of the pollen of the weeds on a large portion of the community, they are objectionable for many other reasons. They are unsightly and depreciate the value of adjoining residential property, and increase their fire risks. They harbor mosquitoes and other injurious insects, and fugous diseases of cultivated plants. Many of the insects that make city gardens a difficult problem, live on weeds, where they thrive and multiply, and make their control almost an impossibility.

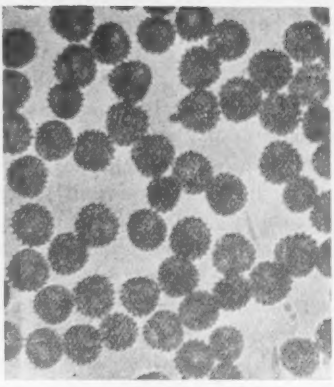
The public welfare demands that whatever is dangerous to life and health should be removed, and this applies as specifically to the hay-fever producing weeds as it does to the control of the sources of typhoid fever and malaria. The aggregate number of suffering and loss of efficiency resulting from hay-fever, affecting as it does, so large a proportion of the entire population, compares with these diseases in importance from a public health standpoint.

The effects of neglected weeds is not only the direct result of this pollen on hay-fever sufferers, but their potential effect in developing new cases. Our investigations in the Asthma and Hay-fever Clinic of the Charity Hospital have shown that in addition to the 37 per cent of the cases that have direct relatives with hay-fever or asthma[4], 28 per cent of their direct relatives, although without hay-fever, show a sufficient reaction to the pollen protein to indicate a distinct allergy. Most of these cases, therefore, require only an excessive exposure to such pollens to develop an anaphalaxis, that will afterwards make them victims of this distressing disease.

Unfortunately, most health officers, except in the few instances in which they are themselves the victim, are not yet educated to the standard where they recognize the importance of the control of hay-fever weeds. With the growing education among the public regarding the cause of hay-fever, however, the demand for relief by the large number of hay-fever sufferers is asserting itself more and more. The public has already begun to resent, in the enforcement of weed laws, a laxity that saves a few dollars to absent owners, but subjects thousands to suffering from their neglect.

GEOGRAPHICAL DISTRIBUTION.

The form of hay-fever and its seasonal development in the different states naturally depend upon the hay-fever plants which grow in their soil. These may vary not only in the different states, but sometimes even considerably in the same state.



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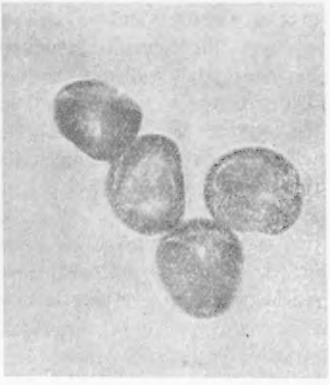
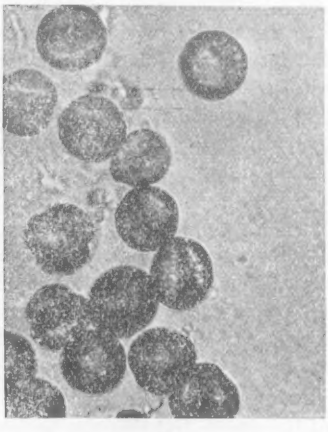


2

1. Pollen of important hay-fever weed. Common ragweed, Ambrosia elatior, x 500 diameter. Principal cause of fall hay-fever in the United States.

2. Pollen of important hay-fever weed. Johnson grass, Andropogon halepensis x 500 diameter. Common cause of summer hay-fever from New Jersey and Pennsylvania to Kansas and Arizona, south to Florida and Texas.



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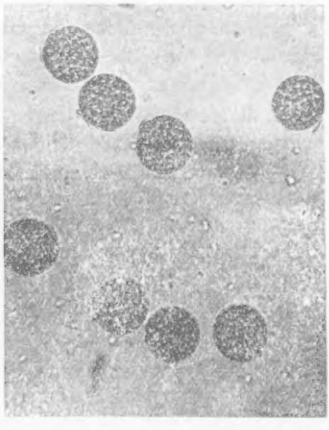

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3. Pollen of important hay-fever weed. Mugwood, Artemisia heterophylla, x 500 diameter. Common hay-fever weed in the Pacific and Rocky Mountain States.

4. Pollen of important hay-fever weed. Goosefoot, Chenopodium anthelminticum, x 500 diameter. Cause of hay-fever from Maine and Ontario to Florida and west as far as California.

In the eastern section of North Carolina, for instance, with its greater humidity and milder climate, the fall hay-fever, which here is due to the common and giant ragweeds, may continue as late as November, while in northwestern North Carolina, with altitudes of 2,200 feet and over, the fall hay-fever, caused almost entirely by the common ragweed, usually terminates promptly about October 1.

The state of Washington has a still wider range of climatic conditions, and consequently of hay-fever flora. The rain-fall ranges from 100 inches at points on the coast to 7 inches in the interior just east of the Cascade Mountains, and then increases again to 35 inches in the eastern part of the state. The altitude ranges from sea level to nearly 15,000 feet, and the climate from districts where it rarely frosts to farming sections around Spokane where it frequently reaches 30 degrees below zero. The plants causing hay-fever and their season of pollination naturally attain a wide variation under such different conditions.

With its area of nearly 266,000 square miles, and its diversified surface, the state of Texas also presents marked variations in the plants that cause hay-fever, and in their blooming season. The low prairie lands along the coast offer a different flora from that of the plateau, the southern border of which is about 1000 feet, but which reaches a height of 2000 feet as the ascent continues towards the arid *mesas* of the *Llano Estacado* and the base of the Rocky Mountains. The soil also varies from the fertile lands of the river bottoms and prairies to the sterile sand of the southern desert.

As the result of these variations, the type of hay-fever in northwestern Texas resembles that of New Mexico, while the southern and eastern portions resembles that of Louisana and Arkansas.

INCREASE OF HAY-FEVER CASES.

The records of the American Hay-fever Prevention Association, based on data from every state of the Union, proves that the number of hay-fever cases has materially increased in the last 10 years. The principal causes of this increase may be divided into two general classes: first, apparent; second, real.

The apparent increase is due to the fact that the public has become familiar with the symptoms of hay-fever, and that the disease is not necessarily limited to any special season of the year, and thousands of cases of "summer colds," "acute catarrh" and "rhinitis" are now recognized as true hay-fever. From an immuniological standpoint, this is a valuable asset in public health, as recent cases are much more amenable to treatment than long-standing cases.

The real increase in the number of hay-fever cases are: first, general, and second, local. The first, which is common in all parts of the United States, and which is an important factor in the increase of hay-fever, is the greater facility of transportation. Formerly the masses were compelled to reside near their places of occupation, and usually in cities, where the congested population left little room for vacant lots overgrown with hay-fever weeds. With the advent of the electric cars, automobiles and local trains, a considerable portion of the population lives in the suburbs, the majority of which are more or less infested with hay-fever weeds.

The local causes of hay-fever vary in different sections of the United States. In some localities, extensive forests, with few weeds, have been cut down, many of which are being used for agricultural purposes with their attendant weeds. Many of the smaller towns are surrounded with farms that are no longer productive, and have become overgrown with weeds.

In the neighborhood of some of the southern cities, large areas have been reclaimed from swamps and marshes, which were practically free of hay-fever weeds, and a large proportion of these, on account of their rich fertility, have become a perfect jungle of hay-fever weeds, the giant ragweed being one of the most common. In some places, these weeds attain a heighth of 10 to 15 feet, and their noxious pollen is carried over miles of the surrounding territory to the great distress of hay-fever sufferers.

In other sections, arid land has become available for agricultural purposes through irrigation, and extensive areas have been placed under cultivation, and here again the parasitic weeds appear, a source of expense to the farmer and a menace to health.

The principal causes here enumerated, have resulted in so large as access of hay-fever cases, that it merits the earnest attention of legislators and of city and state boards of health, and of the United States Department of Public Health.

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THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

(Continued from page 482)

Correction of Nasal Deformities by the Intranasal Method. Dr. L. P. Berne.

DISCUSSION.

DR. CARTER congratulated Dr. Berne on the beauty of his pictures and the graphic manner in which he had presented the subject. He differed with him, however, in his methods. In the first place he preferred to have his patient under a general anesthetic; in the next place he uses an autogenous transplant composed of conjoined cartilage and bone taken at the time of the operation from the patient's rib. The transplant is introduced from within the nose and no scar results. The transplant becomes an integral part of the framework of the nose; it is never absorbed, when properly introduced, it remains alive and grows, its growth being regulated by the functional demands made upon it. In Dr. Carter's work the nose is restored to its histological and physiological entity in addition to its restoration from a cosmetic point of view.

It has been shown long ago that the tissues do not long tolerate the introduction of foreign bodies and Dr. Carter does not approve of the use of ivory, as advocated by Dr. Berne, celluloid, vulcanite, etc., all of which have been thoroughly tried out by some of the ablest operators.

Dr. Carter devised the methods he now uses about fifteen years ago and a number of cases have been X-rayed from time to time by Dr. Law. Serial plates made from time to time for many years after the operations show that the bone transplants are in perfect condition. These plates show also that where the transplant had a considerable amount of work to do in correcting the deformity, that it had grown considerably in order to overcome the resistance; this result could not have been secured by a foreign body, nor even by autogenous cartilage alone, as cartilage remains indefinitely in the tissues in the same condition as when it was introduced.

The tissues should be widely dissected up over the nose and sometimes the cheeks, before the transplant is introduced. Where there is not much support to the tip, Dr. Carter uses a strut of cartilage introduced between the columellae to support the end of the dorsal transplant.

In regard to humps and the other deformities, Dr. Carter has used for a number of years practically the same technique described by Dr. Berne.

The correction of nasal deformities is work that requires a special quality of skill and experience and it should not be attempted by those not possessing these requisites.

DR. SHEEHAN: This is a very admirable demonstration of the work Dr. Berne has been doing. Surely the demonstration was full of animation. As Dr. Berne knows I am against intra nasal procedures, first on account of the certainty of infection that will take place in some cases, and then again the reaction is much greater than by employing an external pathway. By the external route I do not mean the infra-glabellar nor the inner canthal nor the alar nor the horizontal tip end incision of the Albee but an incision made throughout the length of the columella. (Demonstration on black board.) This incision was described to you last year. (See Laryngoscope, September, 1922.) If the nasal septum is to be removed this can be nicely accomplished through the columella incision before the nasal deformity is corrected. I feel there is a decided advantage in using this incision of (Sheehan) as all of the work can be performed in one sitting. By using this incision one is able to work in a sterile field besides having better control of the transplant. The reaction is not very great from undercutting and in due course there is very little if any scarring.

Dr. Berne also knows I am against the use of any foreign substance in the nose for the purpose of acting as a support, whether the foreign substance be ivory, silver filigrees, celluloid or hydro carbon. The French gave these a thorough trial and years of experience have taught them that it was only in the exceptional case did the foreign body stay in the tissues undisturbed over a period of years. The tissues do not take kindly to foreign substance and it means sooner or later expulsion, through infection, abscess formation or what not. The point which had most interest for Dr. Sheehan in Dr. Berne's presentation of his work was what he said about transplants. He quite agreed with the essayist what he had said about the use of the middle turbinate. I shall even go further and state that the middle turbinate bone will either absorb a degenerate into a fibrotic mass in 100 per cent of cases where it is used. If a fibrotic mass forms this may act as a support, but it is not bone. I am not in accord with what Dr. Berne states has been his experience with the use of septal cartilage. It will absorb in all cases where it is used and more readily than the middle turbinate bone. Septal cartilage unlike chrondal cartilage is composed principally of yellow elastic fibres and when separated from its post always absorbs.

It was indeed gratifying to me to hear Dr. Berne state that he uses chrondal cartilage in his severest forms of depression and that he had absolutely discarded the use of bone in any form to act as a support to the nasal bridge. Bone is being hurriedly given up today by most operators as its ultimate fate is still quite a question. The men who formerly vigorously defended the use of bone, have now given it up and are using exclusively chrondal cartilage. It is very interesting to watch the pendulum swing to the safest of all nasal transplants. I never had any doubt but that this change would occur as soon as surgeons became more familiar with its use.

Such leaders as Ferris Smith, whom you have just heard tonight, and who, by the way, has done as much, if not more, constructive plastic surgery in America since the war than any other operator, uses nothing else in this sort of work. And may I be permitted to name such other leaders as Le Maitre of France, Alexandro of Italy, Gillies and Kilner of England; Ivy, Blair, Kazanjian and Staige Davis of America, along with a host of other eminent operators. I personally can see no advantage in using a combination of cartilage and bone, for that part of the bone extending into the soft parts, will become thin, atrophy and finally lose its power to act as a nasal support.

It may be of interest to you, gentlemen, to know that we are carrying out a series of experimentations with bone, and I shall be happy to report to you at some future time, if it is your pleasure, the results of our experimentations. In the event we should find out as a result of our experimentations that bone will live when transplanted to the nose, I have very grave doubts whether I would use it, as cartilage has so many advantages over bone.

Cartilage will neither shrink nor enlarge if its perichondrium is removed. The general opinion amongst plastic surgeons is that chrondal cartilage is an ideal supporting substance for nasal transplantation. It will also live when in contact with bone at one end or both ends, being bound by fibrous tissue. Cartilage is flexible and thus less liable to subsequent fracture, as bone is. It can be easily handled, shaped and whittled to any desired pattern, even handling the graft roughly does not reduce its vitality, as in bone. If it becomes infected and treated in a proper surgical way the graft in most cases will be saved without materially damaging the transplant. This is not true with bone, it must be removed as a rule. It is just as accessible to obtain as bone. The perichondrium must be removed, as it will cause the graft to curl up, with it occupying its concavity. Twice the amount of cartilage is taken as need be, the excess being refrigerated, so to speak, beneath the skin of the chest. This is done in the event the operation be not successful. Under local anethesia, the graft can be removed and again reinserted in the nose. This is not true of bone, for if you inset it, in loose tissues it

will gradually absorb. It seems cartilage gives better cosmetic results. Autologous cartilage grafts never absorb if the graft successfully takes. Cartilage should always be transplanted free. Cartilage thoroughly keeps its shape, although held loosely by fibrous tissue. Cartilage grafts may be exposed when taken from its host for hours. This is not so with bone; to continue viable it must be transplanted immediately. No bandaging as a rule is necessary when using cartilage. In the case of the bone transplant, it is necessary to use a tight bandage from 7 to 10 days in order to keep the bone in situ. If one uses cartilage as a transplant the airway is as a rule never clogged. In the case of bone the airway is clogged from two to four weeks as a result of tight bandaging. If bone is used it makes a difficult job for one, as the reaction from subcutaneous undercutting is tremendous; the chiseling into the bone of the forehead is tedious, time consuming and one is liable to mutilate the periosteum, as it is no easy job to separate the periosteum in this area, when one is practically working in the dark.

DR. TIECK said that he had been invited to discuss Dr. Berne's work and had greatly enjoyed seeing the pictures. The surgical procedure was the same that he himself had been employing for 14 or 15 years, concerning which he had written and showed the pictures at the 10th International Congress at Paris last summer. As for the foreign body transplants, he had employed gutta percha, silver plates, very hard paraffin, etc., and has made moulds on the outside of the nose of paraffin, and gutta percha, so that the nose could be shaped exactly as desired, then made the incision on the inside of the nose and introduced the would be transplant and sewed the wound up again. They hold for a number of months and some for a longer time. One transplant of white gutta percha was in the nose 15 to 18 months and came out only five weeks ago. These foreign bodies do not hold forever, they will stay in for a while as long as you are surgically clean. When they come out they leave a cavity filled with granular tissue. Then the question arises what should be done with this cavity to prevent further sinking of the nose. In this cavity I have transplanted fat with a good result.

So far as the pictures are concerned, they may be very deceptive. Dr. Tieck said that he had been doing this work for 15 years and had treated many cases, and had had some bad results which he is ashamed of today. On the other hand he has had many splendid results, but the pictures amount to nothing unless one can show the patient after two or three years.

I am still using middle turbinal bones in cases where small transplants are needed and find them very satisfactory. At least 50 per cent of them hold and the transplanting can be accomplished without any destruction and without great surgical interferences.

DR. HARRIS said that he could only repeat what Dr. Sheehan and Dr. Carter had already said about the admirable presentation of the subject by Dr. Berne. Aside from any controversial question it represented an immense amount of work, for which he was to be complimented. Every one should say amen to such work as he and Dr. Carter and Dr. Sheehan are doing.

Referring to the value of artistry, Dr. Harris said that such work is not pure mechanics but that the artist counts tremendously in the results attained.

Correction of Nasal Deformities by Intranasal Method. Illustrated with Motion Photography, Animated Pictures and Slides. Dr. Luis P. Berne.

To be published in a subsequent issue of THE LARYNGOSCOPE.

DISCUSSION.

DR. BERNE replying, thanked Drs. Harris, Carter and Sheehan, for their splendid expressions regarding his efforts, and particularly regretted Dr. Tieck's somewhat unkindly remarks.

In answer to Dr. Carter's remarks referring to columella support, he desired to call attention to the cases shown on the screen, where there

was a loss of the columella and where correction was brought about according to the method devised by Dr. Gillies of London, and elaborated upon in this country by Dr. Sheehan. In such cases costal cartilage denuded of perichondrium, excepting at the knee and the frenal end, is used in sufficient length to include the dorsum of the nose, and the columella.

Why use general anesthesa when all the results can be obtained painlessly under local anesthesa, besides the operator can better judge the progress in the changes made, when the patient is awake than when the muscles are limp and the face expressionless.

Dr. Berne said that he does not employ bone as transplant because in his opinion the best advantages are found in cartilage, costal or septal, and ivory.

Does Dr. Carter mean to convey by elasticity of the skin, the overcoming of any tension which may occur after the transplant is inserted, if so it is true Dr. Berne said that he undercuts beyond the dorsum of the nose.

Dr. Berne stated that he had definite reasons why he does not use the extranasal route, (1), The patient's objection to the external incision; (2), The scar which ineviably follows, especially due to trauma from instruments; (3), If the nostrils require correction also, too many incisions about the tip would inhibit healing; (4), The impossibility of correcting a bulbous nose through an external opening; (5), No infection by intranasal method in over three hundred cases.

Dr. Berne agrees with Dr. Sheehan that septal cartilage absorbs, but in its absorption it leaves a fibrous deposit which is sufficient to fill in the slight depression wherein it is used.

Dr. Berne reiterated that he only employs ivory in properly selected cases but always prefers the transplanting of cartilage.

It may be true said Dr. Berne that Dr. Tieck has been correcting nasal deformities for many years, but here is where Dr. Berne claimed priority for his plastic surgery of the face took root when as a mere boy he evinced an aptitude for sculpturing, drawing and painting.

Dr. Berne did see Dr. Tieck operate, as well as he has observed many rhinoplasts both in our own country and abroad, but he had learned one thing and that was not to use Dr. Tieck's technique.

Dr. Berne wished to know where Joseph, Gillies, Lemaitre, Sebileau, Roberts, Roe, Joseph C. Beck, Iglauer, Lee Cohen, and a host of other eminent surgeons obtained their knowledge of rhinoplasty?

In Dr. Tieck's contribution to this subject nothing was ever mentioned by him of the correction of the soft structures of the nose.

Rhinoplasty, said Dr. Berne, demands in addition to a high degree of skill, an appreciation of the artistic nature of the task.

Dr. Berne presented three patients, one operated upon two years ago for a humped nose, a second case that of a transplant of ivory inserted eighteen months ago and a third case of cartiiaginous transplant of one year duration.

SECTION ON OTOLOGY.

March 1, 1923.

Brain Abscess. Dr. E. B. Dench., New York City.

The development of a brain abscess is characterized by two distinct types of symptoms; first, those due to general infection characterized by a rise in temperature, an increased white cell count and an increased polynuclear count; secondly, by symptoms due to the fact that the lesion is in the cranial cavity. Chief of these is headache, then follow symptoms dependent upon the particular portion of the brain involved and these are known as localizing symptoms. The symptoms due to acute infection are frequently of very short duration; that is most of the cases of brain abscess are seen in what is known as the latent stage.

The stage of invasion characterized by high temperature, and rapid pulse may last only a few hours, the temperature then may fall to normal or sub-normal and remain so indefinitely, while the pulse from being rapid quickly becomes slow. The headache, always a prominent symptom of brain abscess is persistent and is one of the most valuable diagnostic symptoms. Vomiting is also observed in fully 50 per cent of the cases and is more common if the abscess is located below the tentorium than if it is located in the cerebrum. My own statistics show that the abscess occurs in the cerebrum much more frequently than in the cerebellar, the proportion being about four to one. The statistics of Pitt, based on post-mortem examinations, however, show that abscesses of otitic origin occur more frequently in the cerebellum than in the cerebrum, but I think that he is the only author who holds this view.

Invasion of the brain substance may take place either by direct extension or through the blood current. Extension by contiguity of structure is the most common method in my experience, by which the brain substance is invaded. When the cerebellum is the site of the abscess, invasion very frequently occurs through the internal ear and occasionally by direct invasion through the lateral sinus, although the labyrinth is by far the most common avenue of invasion.

If a cerebral abscess of otitic origin is located upon the left side its recognition is comparatively simple, as these patients almost invariably suffer from a form of aphasia in which they are able to name the use of an object shown them, but are not able to name it; a purse is "something to hold money in," a match "is something to light a fire with," and a key "is something to open a door with." This form of aphasia is so characteristic that when present it makes a diagnosis absolutely certain, and locates the abscess in the first or second temporal convolutions. I have seen one case of "jargon aphasia" where the abscess was in the Isle of Reil, but in all of the other cases the aphasia has been of the type mentioned.

If the abscess is situated in the right tempero-sphenoidal lobe one must depend upon the persistent headache, the slow pulse and the absence of localizing symptoms in arriving at a diagnosis. When the abscess is located in the cerebellum, the subjective vertigo, the staggering or sprawling gait, the vomiting and the headache are the symptoms which lead us to make a diagnosis. The occurrence of symptoms suggestive of cerebellar abscess in conjunction with a dead labyrinth naturally go far to confirm the diagnosis. In cases where cerebellar infection has taken place through the labyrinth, the nystagmus toward the affected side with a dead labyrinth renders the diagnosis of a retro-labyrinthine condition certain. One sign, common to all cases of increased intra-cranial pressure, must be looked for where intra-cranial suppuration is suspected, I refer, of course, to "choked disc." While this, of course, is not characteristic of brain abscess, but simply is an evidence of increased intra-cranial pressure, its presence with other symptoms of abscess helps to confirm the diagnosis.

Just a few words about the results of treatment. MacEwen reports 19 cases of cerebral abscess, not all of which were of otitic origin, of these 18 were cured, remarkably favorable statistics of such a grave condition. Korner collated 55 cases, with 29 cures. My own cases number 24, with 14 deaths and 10 cures. Of 100 cases of cerebral abscess which I collated 52 were cured and 48 died.

When we come to cerebellar abscess MacEwen reports 4 cases all of which wre cured. In the cases of brain abscess which I myself have operated upon 5 were cerebellar and all of these died, although 1 lived three years after the operation. Of 102 cases of cerebellar abscess collated by myself 69 died and 33 were cured.

In the treatment of brain abscess much discussion has arisen as to te site of election for opening the abscess. Cases which end fatally

after the abscess has been opened, die either of meningitis or of encephalitis. Most operators have felt that if the arachnoid space could be shut off, the chances of meningitis following operation would be very much lessened. For this reason many operators have preferred to open the abscess through the infected area and this should aways be done if the exact avenue of infection can be determined. Of the cases of cerebral abscess collated by myself 41 were opened through the infected area. Of these 27 were cured and 14 died. 37 were opened through the squama, a sterile field, and of these 18 were cured and 19 died. In 22 cases the method of operation was not known and of these 7 were cured and 15 died. My own feeling has always been that where the route of infection can be clearly followed the abscess can be more safely opened along this route, but where the path of infection can not be demonstrated that a prior decompression operation with incision of the dura and a packing off of the arachnoid space, deferring the opening of the brain substance for 12 or 24 hours would be the safest procedure. Unfortunately, I have not been able to carry out this procedure in a single instance.

The method of Lemaitre in entering the abscess first with a needle and then inserting a drainage tube of almost capillary size, increasing the size of the tube daily until it is large enough to afford good drainage has yielded good results in his hands. I have seen the tubes and needles used by Lemaitre and must confess that I hardly see how he is able to introduce even the smallest of his tubes through the opening in the dura. The results claimed by this procedure are, however, excellent.

In cases of cerebellar abscess, the abscess may be opened either in front or behind the lateral sinus or through the lateral sinus. Of 102 cases of cerebellar abscess collated by myself 45 were opened behind the sinus, 25 cured and 20 died. 11 cases were opened in front of the sinus, 4 were cured and 7 died. In 46 cases the method of operation is not stated and of these 4 were cured and 42 died. The region of the lateral sinus is naturally the ideal one for drainage, but unless the sinus has been the avenue of infection I hardly think it wise to attempt to obliterate it in order that the abscess may be drained through this region, although this procedure is advocated by many excellent operators.

DISCUSSION.

Dr. Foster Kennedy said that as one of the speakers was to talk about meningitis and another about brain abscess there was but a limited field left for him to discuss, but that the Chairman had suggested that he give his opinions as to the symptoms of impending cerebral complications of ear disease. To him the most significant symptom of the onset of a brain abscess in intermittent stupor. There is, however, a stage in which stupor exists and in which no abscess exists—a stage in which there is a progressive softening of the tempero-sphenoidal without the formation of pus; and the exploration of the brain before the formation of pus, when the tempero-sphenoidal lobe in undergoing softening, will be of no use. The proper procedure regarding the work of operating in that pre-abscess stage is not yet quite clear, but operation should be postponed until there is evidence of an expanding lesion. It would be interesting to have Dr. Elsberg's opinion as to whether a decompression operation in that pre-abscess stage is advisable, is a point which such a meeting might very well try to determine.

What is the symptomatology of tempero-sphenoidal lobe abscess—taking that as the lobe most commonly affected through the middle ear. The tempero-sphenoidal lobe is a vast, largely uncharted field. On the right side it is the so-called latent area in the brain; therefore its symptomatology—the picture produced by its invasion by an expanding lesion—requires some thought. The most common symptom of an expanding lesion in the tempero-sphenoidal lobe is stupor; the next is slight involvement of the pyramidal tract, etc. It is the first symptom which

will appear. It will appear in a particular way, owing to the position of the tempero-sphenoidal lobe in regard to the motor area; there will be an inequality in the hemiplegia produced. The weakness will be more marked in the face than in the arm, and more marked in the arm than in the leg. This is characteristic of pressure on the pyramidal tract, coming from the tempero-sphenoidal lobe and acting indirectly from below.

The next symptom, if the expanding lesion be found to be placed, particularly in the tip of the lobe, will be the so-called incinate—discharges of taste and smell, etc. That must not be confused with the evil taste produced in septic conditions. A differentiation can be made between the two and by finding whether the unpleasant taste is accompanied by an unpleasant subjective sensation of smell, also whether that be parorysmal or fleeting.

The next symptom of importance is aphesia—when the expanding lesion occurs more posterior in the lobe.

There is another symptom and that is the voluminous mental state, the dreamy mental state, the typical tempero-sphenoidal epilepsy, which may not be associated with convulsions but which consist in the appearance of two kinds of attacks—one, the major attack; and the other, a minor. The major attack often consists of hallucinations. Hallucinations of taste and smell have already been referred to, but these hallucinations are not merely subjective. The hallucinations of the tempero-sphenoidal fit are highly constructive; they are exceedingly complex, not true sensory phenomena. The hallucinations of this lobe will make a man see flashes of light coming from the sky and he will see objects of such light; but what is meant by saying hallucination of the tempero-sphenoidal fits is that you will get people who see scenery, trees, people, etc. One patient with tempero-sphenoidal abscess always saw a woman at her left hand side who spoke to her, whom she though she recognized though she could not remember who she was nor could she remember what was said. This spectre was always associated with that condition. That kind of constructive hallucination sometimes accompanies hallucinations of smell, etc. One patient spoke of an old woman dressed in rags, etc., which was simply an association of one subjective irritation joined on to the very highly visual condition of the tempero-sphenoidal state. So that on the right side you have a symptom capable of identifying the tempero-sphenoidal lobe under attack.

Dr. Foster said he had not mentioned the minor stage but the major stage, which consists of highly complicated visual phenomena; the minor voluminous state is that which the French call the sensation of deja vu—which we have all had—the sensation of having gone through that particular sensation before; as though a veil were drawn away and some knowledge transcendentally obtained, and also definitely associated with great fear, and which is usually an epigastric sensation of fear. That may last a very short time, and is of gastric origin. That is not to say that such minor attacks cannot develop without a gross organic lesion, but when an organic lesion is found in the brain and such phenomena develop, one may be quite sure that it is in the tempero-sphenoidal lobe.

A word about the aphasia, when the speech center is attacked. That seems to depend upon heredity, whether they come from right-brained stock or left-brained stock. The aphasia is of a very characteristic type; it is usually a simple anomia, simply inability to recollect names, to quickly give the noun for the object seen; the retention of all other parts of speech, verbs, adjectives, etc., but a distinct deterioration in the naming faculty. That is the characteristic of the aphasia connected with the tempero-sphenoidal lobe. The abscess formation from ear disease is practically always associated with a certain amount of meningitis, and nearly always you will find a stiffness of the neck.

DR. C. A. ELSBERG said he had had some very interesting and very peculiar experiences with meningitis as a complication of ear disease, one of which he wished to refer to. Year after year he has the striking experience of seeing patients whose ear drums have been incised by the otologist, soon after which—often within a few hours—they developed meningeal symptoms. Those were usually fresh air infections and in a very few hours the patients developed meningeal symptoms, usually with a fatal outcome. He did not mean to say there is a certain connection between the myringotomy and the meningitis, but the occurrence was so frequent that the otologists ought to take cognizance of it, and should consider whether the incision of the drum membrane at too early a stage might not favor the very complication it is desired to avoid.

As to the treatment of meningeal complications, Dr. Elsberg said his results were practically nil. Many years ago he had tried drainage of the spinal canal, and washing out of the subarachnoid and ventricular systems. A few patients with meningeal symptoms and changes in the lumbar puncture fluid had done well, but they were patients in a very early stage, called by some meningismus, and who might have gotten well anyway if nothing had been done. The results in the rest of the patients were absolutely nil—every single one succombed to the disease.

Regarding brain abscess, Dr. Elsberg stated that Dr. Dench had covered the symptomatology very thoroughly, but Dr. Dench had spoken only of the acute abscess, the variety which the otologist sees within a few days of an acute ear infection or an operation upon the mastoid or sinus thrombosis. Dr. Elsberg stated that he had seen a considerable number of patients with this acute abscess, but he had seen many with intra-cranial suppuration months or years after the ear disease or after the occurrence of a chronically discharging ear. Many of these patients did not have a single sign of increased intra-cranial pressure, but mainly the signs of destruction of portions of the brain. Some of the patients had very slight evidence of intra-cranial suppuration and a few had died suddenly, the abscess being found only post-mortem. The problem of the treatment of abscess of the brain complicating ear disease depends upon the character of the abscess. The acute abscess may either have from the beginning a good lining membrane which isolates it from the surrounding brain or the walls of the abscess are formed by infected and degenerating brain tissue. In the former variety the abscess within the lining membrane enlarges and does not directly involve the brain tissue unless a rupture occurs. The second form of abscess increases in size by a progressive degeneration and sloughing of brain and this variety usually ends fatally no matter how thoroughly the abscess is drained.

The abscess that runs a chronic course is usually one that has a good lining membrane which shuts it off from the surrounding brain tissue. If such a chronic abscess increases in size, it may cause a marked increase of intra-cranial pressure and all the signs of an expanding neoplastic lesion. The more acute forms of brain abscess may of course also cause increased intra-cranial pressure either by its presence or as a result of swelling of the infected brain tissue.

Dr. Elsberg then demonstrated on the blackboard the physical differences between the abscesses that have no lining wall and those that have a well marked lining membrane, and he showed how difficult it was to satisfactorily drain the abscess whose walls were formed of sloughing brain tissue while the drainage of the walled-off abscess can be very satisfactorily accomplished. The one principle in the surgical treatment of brain abscess is to open the abscess without disturbing surrounding brain tissue and to fix the drainage tube so that it will not be disturbed and will not injure the wall of the abscess either at the time of the operation or in subsequent dressings. Only a small incision should be made in the dura and when the aspirating needle is in the abscess cav-

ity, small packings soaked in iodine should be used to wall off the meninges. A grooved director is then passed into the abscess cavity along the needle and the needle then withdrawn. The tube is then inserted along the grooved director and when pus escapes through the tube, the grooved director is withdrawn. Part of the rubber tube outside of the scalp is then split in half and each half fixed to the scalp by a silk suture. Over this the dressings are applied. When the tube is fixed in this way, it can not be pushed further in by the dressings and can not be pulled out when the dressings are changed. The danger of injuring surrounding brain tissue is thus reduced to a minimum. The tube should remain in place until there is absolutely no discharge, and then should be shortened and again fixed to the scalp. It should never be withdrawn from the cavity and re-introduced, but may remain in place for any number of weeks.

Dr. Elsberg stated that with this technic, he believed that much better results could be obtained than with the methods of drainage ordinarily used.

DR. DENCH, referring to Dr. Elsberg's remarks about myringotomy, said that in the cases referred to by Dr. Elsberg, the middle ear condition and the meningitis were probably not inter-dependent, but were concomitant in the same individual. He had written on the subject in 1918 or 1919. It seems impossible, of course, that a very trifling inflammation of the middle ear can cause a meningitis which would be fatal within twenty-four hours after the inception of the acute otitis. No one would dream of benefiting the meningitis in the least by incising the drum membrane in a case of this kind. It seemed to the speaker a pity that occasionally an enthusiastic otologist would insist on operating on a case of this character and then report it as one of fulminating meningitis secondary to a mild otitis. Most of the cases so reported were cases of concomitant meningitis.

DR. GLOGAU asked Dr. Dench if in some cases of meningitis following acute middle ear suppuration we might not have to deal with congenital dehiscences in the tegmen of the tympanum or the antrum. Besides we might have to deal with a persistent fissura petro-squamosa which permits the invasion of pus from the middle ear to the meninges, even in apparently mild middle ear suppuration.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

March 28, 1923.

Acute Glossitis with Spontaneous Rupture. Dr. Saul Knopf.

The patient was admitted to the Clinic of Dr. Harmon Smith at the Manhattan Eye, Ear, and Throat Hospital on February 19th with a history of difficulty in swallowing and impediment of speech, and with the tongue practically protruding from the open mouth accompanied by dripping saliva. The family history was negative. Four days prior to admission he complained of pain in his throat and had some difficulty in swallowing. His family physician thought he had a sore throat and gave him appropriate medication, but it failed to relieve the condition. A few days later he was referred to the clinic and came under observation at the Hospital. He complained of severe pain, difficulty in swallowing and in talking. There was a slight degree of tenderness in the deep cervical glands; temperature 102.5; pulse 90. The patient looked quite sick, and presented the picture of a typical peritonsillar abscess. His tongue, however, was red and leathery looking, suggesting erysipelas, and filled almost the entire buccal cavity. His throat looked all right. the tonsils and pharynx were normal, but nothing could be seen beyond the pharynx. he complained of headache, pain in the back of his neck, and a feeling of general malaise.

Dr. Knopf said he reported the case on account of its rarity, although hundreds of throat cases were seen at the Manhattan; and at Bellevue he had not seen a single case of the kind in six or seven years. Considering the fact that so many injuries of the buccal cavity and tongue occur, it was remarkable that more cases of this kind were not seen. Dr. A. B. Bennet of Washington had a series of cases extending from 1860 to 1906, and had only 145 of these cases. The mortality was very low, only 4 died. Most of these cases had spontaneous rupture.

The etiology has been given in many cases as injury by toothpicks or pins, or perhaps biting the tongue during an epileptic seizure, but this seems hardly probable. It is likely most of the infections have been due to carious teeth. The lesion was mostly toward the dorsum and back of the tongue, rather than to the front. The usual symptoms are those described in this case; and one might think of a cryst, or a gumma, or tuberculosis, or carcinoma.

When this patient was admitted to the hospital the tongue felt soft at the apex, but going toward the back it was thick and leathery to the touch, and toward the dorsum of the tongue there was a marble-like feel, which suggested that as the location of the abscess. Most of these cases I believe are infection of the circumvallate glands. Although more or less of them are of an infectious type, they are seldom fatal, and often resolve by themselves. They are usually unilatral.

This patient was sent to the hospital for observation and treatment, for twenty-four hours, because it was not deemed advisable to make an incision. He was kept under treatment with frequent hot irrigation and ice-bags to the throat. A few hours before he was posted for operation, he felt a large quantity of pus in his throat, spat it up, and then had rapid relief.

Most of these cases open up in the posterior portion around the area of the lingual tonsil. After the rupture of the abscess, examination back of the tongue revealed a large quantity of hypertrophied tissue from which a small amount of pus was exuding. This occurred on the 20th of February. Since then the man had improved rapidly.

DISCUSSION.

DR. MAX UNGER testified to the rarity of the condition, and said he had seen only one case in private practice, twelve years ago. The history was that of sudden onset of pain in the tongue and which had lasted for two days. When he first saw the patient the symptom which pointed to the tongue as the seat of the disease was the pain caused the patient by a tongue depressor. The tongue was not much enlarged; but on palpation a small tumor was found at the base of the tongue in front of the left anterior pillar. This was incised through the side of the tongue and one or two cubic centimetres of pus were evacuated. The wound was packed, but the packing fell out in a few hours and the condition healed without further treatment.

DR. FREUDENTHAL said that these cases of abscess of the tongue are very dangerous, although the mortality may appear very low. As soon as one makes the diagnosis of abscess of that region, whether at the base of the tongue, or of the epiglottis, the patient ought to be kept under constant supervision. He then told of a case which happened many years ago, when a young woman patient left him and went to her physician, who told her he would see her the next morning, but the next morning she was dead. The abscesses of the epiglottis are the most dangerous, and in such cases a tracheotomy should be provided for at once. The most significant symptom is the pain when a tongue depressor is used. It is often difficult to know where to make the incision, but sooner or later it can be found out, and the incision once made the danger is over.

DR. E. ROSS FAULKNER: Dr. Faulkner said he remembered two cases of abscess of the tongue, which he had opened externally under local anaesthesia. They were in the center, and produced quite alarming

obstructive symptoms with high temperatuse. It is a very simple matter to open them in the center line, as there are no anatomical structures of importance. A suppurating Ranula will give symptoms very similar to a lateral abscess in the tongue, and probably the two conditions are often confused. It seems probable that abscess in the center may be due to suppuration in the Thyroglossal tract.

DR. LEDERMAN cited four cases of abscess at the base of the tongue which he had reported to the Section four years ago, and which at that time he called phlegmonous epiglottidis. It is a peculiar fact that these cases of edema do not extend to the internal surface of the epiglottis on account of the texture of the perichondrium but is limited to the external surface. You have the edema, however, and it may extend into the larynx. The cases referred to were all incised and the patients fortunately recovered without any unpleasantness; but such conditions do give alarming symptoms and one must be ready to do a tracheotomy if necessary. Not long ago he had seen a case which, while not germane to the discussion, the lesion involved the tongue. The patient, a male, was a hemophiliac who came to the office unable to speak distinctly; his tongue was three times the natural size, and black resembling the tongue of a parrot. It was a most astonishing condition and the patient was very uncomfortable, for the tongue was so large that it interfered with respiration and deglutition. He was given adrenalin, internally and locally and ice applications externally and internally. In twenty-four hours the tongue became consderably reduced in size. Knowing the man to be a hemophiliac, Dr. Lederman did not wish to incise the tongue or perform a tracheotomy unless urgent symptoms arose, for he had seen him some time previously suffering from a submucoid extravasation of blood. As this hemorrhage had gone down the larynx and on both sides of the pharynx it looked like the outstretched fingers of the hand. The patient at that time was kept in an institution and watched for two weeks.

DR. IMPERATORI said he had seen several cases in the past year or so, with the same findings as that reported. All of these cases had followed drinking bouts and all had severe pyorrhea alveolaris and edema of the epiglottis. All of these cases recovered.

They were treated with a 2 per cent warm sodium citrate solutions, as an irrigation, administered every two hours.

DR. KNOPF remarked that an important point was that the abscess occurred on the dorsum of the tongue posterior to the medium line. It would be well if a patient should come in with difficulty and pain in swallowing and no peritonsillar abscess was found, to keep in mind the possibility of abscess of the tongue.

Prolapsed Ventricle of the Larynx. Dr. Knopf for Dr. Harmon Smith.

The patient was first seen by Dr. Smith at the clinic, complaining of hoarseness. He had no pain nor difficulty in swallowing—only the hoarseness. He stated, however, that he worked in a place where he had to shout in order to be heard, and about the same time he caught a cold and had an explosive cough like a bark. When examined he was found to have a mass which seemed to be protruding with perfect evenness along the false cord and overrode the true cord. The chest examination was negative, and a provocative Wassermann test was negative. Some of the men thought it was a prolapse of the ventricle, but there was some uncertainty, and finally it was decided to treat him tentatively with K. I., but he disappeared from observation. Finally he was traced and induced to come to the meeting tonight. The mass was practically half the size now seen. The condition instead of being on the entire level of the cord was simply on the anterior half. The case was presented rather as a matter of diagnosis, rather than as an actual prolapse of the ventricle.

Two Cases of Frontal Sinusitis (Radical Killian). Dr. Francis W. White.

T. L., 15. The patient was received in Dr. McCullagh's Clinic at the Manhattan Eye, Ear, and Throat Hospital, with the following signs; Redness and swelling of left upper eyelid with edema over the frontal region of the same side, but more particularly toward the median line. Slight tenderness over this area. The left eye was almost closed. The temperature on admission to the hospital was 103.2°.

Local treatment intranasally followed by removal of the anterior tip of the middle turbinate. The temperature gradually declined and all signs showed improvement. In a day or so the right eyelid became swollen and red. Then there was a sudden rise in temperature to 103.3°, and an increase in swelling over th left frontal with fluctuation.

In the mean time an X-ray had been taken which showed severe involvement of the frontals with septum much to the right of the median line. Left ethmoid—sever involvement. Right—mild. Left antrum—mild involvement. Right—severe. Sphenoids—large, mild involvement.

Operation—The regular incision for the radical frontal operation was made, and after incising and freezing of the periosteum a perforation was found in the bone about 2 mm, in diameter, slightly above and to the right of the supra-orbital notch, and another perforation about half the size of the former was found midway between it and the junction of the left nasal bone and nasal process of the superior maxilla. Both antra were opened.

Erysipelas supervened starting on the right side of the face and spread to the left side in a characteristic manner.

During this attack blood cultures were negative.

Bacterial growth from the sinus showed a short chain non hemolitic streptococci and a micrococci catarrhalis.

This patient is presented for two reasons. First the X-ray findings, and secondly the presence of perforations.

C. D. Five days after an acute attack of coryza the patient was brought to the Manhattan Hospital as an eye case and then assigned to Dr. McCullagh's service later.

There was marked swelling, redness and edema involving the left frontal area, and the eyeball appeared to be pushed outward and downward onto the cheek. Pain apparently was present and pressure over the left frontal area gave rise to much discomfort. The condition had the appearances of a very severe acute fronto-ethmoiditis. Temperature, 101.2°.

X-ray-frontals absent. Ethmoids show severe involvement, especially the left side. Antra show severe involvement. Sphenoids absent.

As soon as the X-ray report was received, a radical external ethmoid operation was performed.

Wassermann—X X weak.

DISCUSSION.

Dr. Emil Mayer said he wished to make a little contribution to the history of the Killian operation, inasmuch, so far as he knew, he was the first to perform this external operation in New York, at the Eye and Ear Infirmary many years ago. The patient came to the clinic with such acute pain that he could not sit quietly but was walking up and down the room. He was examined at once and such extreme tenderness was found that an immediate operation was advised. He said that he could not spare the time from his work and his children needed him, but he was told that if he did not have the operation his children and widow would need him a longtime. The operation was done. Dr. Mayer said he could distinctly recall the surprise of his associates when the bone was opened and the pus welled out, and the feeling of gratification that at least something had been found which was not done before. In those days there was no X-ray to go by nor was anything known about microscopic examination of the pus. The conditions under which we are working today are much happier than those that obtained in those early days.

DR. HARRIS congratulated Dr. White on the excellent cosmetic result obtained in the first case. This was not a suitable time to discuss the Killian operation, although it was interesting to hear that Dr. Mayer performed the first operation of that kind in this country. One should not loose sight, however, of the need and desirability of a good cosmetic result. He recalled another case, and said with confidence that he had yet to find an incision that for cosmetic results equals the Killian incision, as Killian recommended it first. Apropos the value of the X-ray—he cited a case reported by Dr. Johnathan Wright many years ago, in which he kept on drilling until he entered the brain cavity. That should not be possible in these days, and he very strongly endorsed what Dr. White said—that whatever one may think diagnostically of the X-ray a picture should always be taken and studied before any radical operation was decided upon.

DR. DOUGHERTY told of a case in the hospital, referred by the Medical Division with all the symptoms of frontal sinusitis. Upon consultation with the Roentgenologist it was decided that the left frontal sinus required operation, and he started in to perform a Killian operation. He thought at first he was going through the man's head; then tried the other side, and found no sinus there at all. There was an exostosis that filled the whole cavity, and the symptoms were merely reflex symptoms, and both the clinical and the X-ray diagnosis were absolutely wrong.

DR. HURD said that sometimes one does not have the opportunity to have X-ray studies made, and has to work rapidly. About four weeks previously a girl of fourteen or fifteen came to the office with an abscess involving the sinuses. She was very sick, and was sent to the hospital and operated upon at 2:15 that afternoon. There was no time to make a study of the sinuses. He made the triangle of Killian below, got into the anterior ethmoidal cells, and followed the frontal duct up with a probe. She had a frontal sinus three-fourths of an inch by an inch, a flat shell, which was uncovered and obliterated. . . . That was his way of treating such cases. He had seen several cases where they had gone through the typical way, and he had the task of telling the family that they had only a few days to wait and it would be all over.

DR. MCCULLAGH thought the interesting point in this case was the perforation of the anterior wall. All had seen perforations into the orbit. When he first saw Dr. White's patient he was very much puzzled by the picture presented. It was the first instance he had seen of perforation through the anterior wall. Dr. MacKenty had told him he had seen but two. It seemed to be of comparatively rare occurrence.

DR. IMPERATORI called attention to the fact that he had reported a case of spontaneous rupture of an abscess of the frontal sinus, before the Section last spring.

As to the cosmetic results, he always wondered whether the patient would stay well.

At Bellevue Hospital it was the teaching of Dr. Coakley, to obliterate the cavity entirely, by pressing the skin backwards into the site of the frontal sinus.

The cosmetic results are poor, but few of these cases recurr.

DR. WHITE, in closing the discussion replied first to the inquiry regarding the Wassermann reaction, which he stated was reported negative.

As to the perforations, it was the second instance of the kind he had seen, namely the occurrence in the anterior wall of the frontal sinus; the other being in a soldier at Fort Oglethorpe. In the latter patient however, the perforation was situated at the extreme outer angle of the sinus just above the external canthus. Inquiry among conferers elicited the fact that none had seen over two or three cases of perforation of the anterior frontal wall.

Concerning the excellent cosmetic results as noted by Dr. Harris, Dr. White said that he did not think any operator claimed to get constantly

or uniformly good results. He did one thing however, he stated, that probably would not meet with general approbation—namely the use of rubber tubing. One piece was put into the nose from the frontal opening and allowed to protrude for a short distance from the nostril. Another piece was brought out from the external angle of the incision. Both were anchored, and especially the nasal one was additionally safeguarded by means of a safety pin passing through the distal or extranasal end. Irrigation with an antiseptic solution was carrier out, the edges of the wound having been sutured. In this manner, pus was kept liquid and flowed out of the lower or nasal tubing, the granulations were kept free from pus, the passage way from the nose to the operative cavity was narrowed thus precluding to a great extent the forcing of infected material from the nose into the operative field. The tubes were changed and smaller ones substituted, the granulations meanwhile filling in solidly the cavity in all directions and giving less chance for marked secondary contraction, which is bound to occur.

Referring to the X-ray study in the second case, and Dr. Hurd's remarks thereon, Dr. White said that through the co-operation of Dr. Law the plates were read while still wet and the absence of frontal sinuses as shown by the plates was of inestimable value. Naturally there are circumstances when a sinusitis may be compared to a malignant appendicitis that is the necessity to operate immediately.

Sarcoma of the Right Antrum. Dr. Irving Wilson Voorhees.

Dr. Voorhees said he had not looked up the literature but assumed that sarcoma of the antrum was not very common—this was the first case he had seen in some years.

The patient was a young woman, 23 years of age, married and had two children, one and five years of age, both well. There was no history of malignancy in the family. The symptoms were rather marked. There was a good deal of bulging into the roof of the mouth and into the right varis. She came in for relief of breathing, as the nose was obstructed and also because she was beginning to have some pain. The X-ray picture showed marked density on the right side of the head, and no definite outline of the sinus. Antrum washing was negative, and the diagnosis of a neoplasm of the right antrum was made. A simple Caldwell-Luc operation was done under local anesthesia. One per cent, novacain and adrenalin was used, the needle inserted under the lip far up until the infra-orbital nerve was reached. Then one cc. of the solution was injected in that region. An injection was then made in the roof of the mouth in the region of the anterior palatine vessels; another in the region of the posterior palatine vessels. The last was made external to the third molar tooth to catch the posterior-superior dental nerve.

The usual Caldwell-Luc procedure was then carried out. On getting in, it was found necessary to deliver the growth by morcellation, as the obstetricians say, for it is impossible to shell it out en masse. The entire naso-antral wall was taken down, and the middle and inferior turbinates were removed. This left a large opening. The cavity was packed with iodoform gauze which was removed the following day. The patient was then referred to the Memorial Hospital for treatment with radium, and had received two exposures about two weeks apart Another exposure would be made the next day. She had greatly improved and was apparently getting well; but, of course, it was impossible to know how permanent the improvement would be.

The growth was apparently attached to the inner wall of the antrum—the naso-antral wall. The pathologist reported it to be a spindle-cell sarcoma. There was very little bleeding during the operation, and the anesthesia was excellent. The whole operation was surprisingly easy, and required only about a half hour. A post-nasal plug was inserted before beginning the operation, but it gave the patient so much discomfort and seemed so unnecessary because of the slight bleeding, that it was soon removed. Suction was used to keep the field clean.

DISCUSSION.

DR. MAYER was very much interested in the case, since we are in a transition stage at present as to just what and how far the surgeon should go, and whether radium or the knife should be used first. Oftentimes the situation is such that the decision rests with the laryngologist patient with an advanced sarcoma of the upper jaw, involving the hard palate and going beyond the median line. It was clear that any surgical procedure in such a case would not be of any great avail, and the and he must follow then his best opinion. He then told of an elderly patient agreed, it was decided to try radium emanations. Dr. Mayer said he had never, excepting in the early days of Salvarsan, seen such a remarkable demonstration of the melting away of that growth from the introduction of something like a dozen small millet seeds of radium emanation. Therefore when considering what should be done in these cases, one will many times be confronted with such another case as he had seen recently, where the sarcoma involved both sides of the nose total occlusion of one side, and where an operation would be extremely bloody; in such a case radium emanation would probably do fully as much good as any surgical procedure.

It was to be hoped that Dr. Voorhees would bring this patient here again later and let the members see her; for it was not so much the temporary effect that is obtained but the lasting effect that means so much as these cases are followed up, and it was to be hoped that later this patient would continue to show the good results now exhibited.

DR. HURD said that Dr. Mayer had brought up the very important question as to the use of radium in malignant sarcoma and carcinoma, and he did not quite agree with him—whether pre-operative, post-operative, or operative. The Doctor had spoken of using radium seed. As he understood the matter, seed are liable to destroy bone, and one might have a necrosis. That was one point. Dr. Hurd said he had several cases in mind. In one case there was a very beautiful result of post-operative radium in carcinoma of the antrum and blocking of the neck; and three years later the patient had a metastasis in the spinal cord, as if the radium had started it up. Another case incidentally brought up the question of local anesthesia. That was the only way to do any antrum operation, but it could be made still simpler by picking up the second nerve. That was used very successfully last winter on a woman; at first a section was removed and Dr. Ewing reported it not malignant; but on going further in at operation a mass of malignancy was found. Enough of this was removed to show to Dr. Ewing, to whom the patient was referred for treatment, and when last heard from the patient had a recurrence.

We seem to be in a stage with radium where we do not know exactly what we are going to do; but it was to be hoped that at the Memorial Hospital they would be a little more frank than they have been so far. In Buffalo they will tell you exactly where they stand. On being asked about radilum in carcinoma of the larynx, they said the results were 100 per cent to the bad, etc., etc.

A report from the Memorial Hospital is not always as definite as one would like to the questions asked: Which is better, the combination, etc., etc. Dr. Hurd closed by saying that in his experience with laryngectomies was that those that did not have radium did better than those that did.

DR. GEORGE D. WOLF told of a case which he showed at this section at the October meeting of 1920. This patient had a Round Cell Sarcoma of the Nasal Vestibule which was probably involving the antrum. This case was not operated on but received intensive radium treatment at the Memorial Hospital. The neoplasm apparently melted away under the influence of the radium. The patient, however, died five months later from intestinal metastasis.

DR. LEDERMAN told of two cases of inoperable malignancy, one involving the rhinopharynx and treated with radium through the nose. One

application of radium caused it to melt away in three weeks. In a case of sarcoma of the tonsil, two applications of radium relieved the growth as completely as if a tonsillectomy had been performed. What the ultimate result would be he did not know, but the effect of the radium in these two cases was astonishing. In the first case the radium was permitted to remain for an hour; a slight burn of the hard palate resulted but caused no sloughing or inconvenience to the patient. Both patients were treated at the Memorial Hospital.

DR. FORBES said that one of the essentials for the successful use of radium was to have the part accessible, and then after removing the growth, planting the radium seeds. It was a great question and one that could not be decided at such a meeting—whether the application of radium before or after operation is the better.

DR. VOORHEES said his feeling was that in future fewer mutilating resections of the upper jaw would be done. He had seen several resections which were frightful to behold and the patients died rather promptly. If one could operate sufficiently to place the radium where desired, as much was accomplished as could be hoped for in the present state of our knowledge. Screened radium was used in this case—not the seeds. As to the method of nerve blocking which Dr. Hurd referred to, Dr. Voorhees said he would like very much to try it. He had always regarded the blocking of the fifth nerve as rather difficult. The procedure in this case is much used by orificial surgeons, and is eminently safe and certain.

All the cases of carcinoma of the larynx that he had seen treated with radium had done badly, and many men of experience do not recommend its use in the larynx at all. He had not seen any cases of sarcoma of the entrum in children and presumed that they were very rare.

DR. J. M. MCTIERNAN presented the history of a patient whom he had under treatment at the Manhattan Eye, Ear and Throat Hospital, in the service of Dr. Harmon Smith:

A child, four years of age, gave a history of having fallen and injuring her nose. Two months later, mother noticing a slight bulging over left side of nose took her to the Clinic. Examination showed the inferior turbinate and lateral wall to be pushed over against the septum. A mass about one and one-half by two and one-half cm., with a distinct outline could be felt over the buccal area. An X-ray showed the antrum and ethmoids to be involved. At a point just within the nostril a section was taken which proved to be a mixed cell sarcoma. Under massive doses of radium externally and needling within the nostril, the mass has entirely disappeared.

Dr. McTiernan knows the present findings are not an end result but reports the history of the case as he believes there has never been a case reported as having existed in a child of this age.

Osteo-Myelitis Associated with Sinus Disease. Dr. E. Ross Faulkner.

Before giving the details of the clinical course of the cases, I wish to discuss briefly the course of inflammatory processes in bone.

Acute inflammation in bony tissue varies in its course as it does in other parts, according to the blood supply of the part, and the density of the tissue. In compact bone, such as the shaft of long bones, the bones of the skull, and the maxillary bones, there is immediate strangulation of the circulation with death of the tissue en masse or necrosis. This dead bone is cast off from the living by a process of ulceration, and the time of separation varies in different regions.

In cancellous bone, such as the ends of long bones, or cellular bone, such as the ethmoid cells and mastoid process, the result of acute inflammation is ulceration or caries. This is accompanied by pus formation, and as the ulceration process extends, an abscess cavity of considerable size may be formed, or various small abscess cavities may form without becoming confluent.

Chronic inflammatory processes in bone result in thickening or sclerosis. The periosteum in all cases is involved in the inflammatory process,

and vice versa. One may state that the bone is involved to some extent in all cases where its covering is the seat of inflammatory change.

The route of infection in acute, non-traumatic inflammations in long bones is through the blood stream, and this may be the method of localization in the bones of the skull and face where the primary phenomena appear in the bone itself. The cases I am going to report tonight, I believe to have been primarily in the bony tissue, with secondary involvement of the adjacent sinuses. If one considers the mucous membrane lining such cavities as the antrum or frontal sinus, one must realize that this membrane is essentially a periosteum and carries on more of the function of the periosteum than the external covering which is especially thin over the bones containing these cavities; hence, with involvement of this bone there is an immediate spread of the process to this inner membrane with resultant pus formation in the cavity.

If one can reason from analogy in other parts, acute inflammation of the membrane covering the bone, either the external or the internal, is not likely to produce a necrosis, but is rather prone to initiate a process which will produce gradual thickening of the underlying bone, unless resolution occurs quickly. Repeated attacks or a single unresolved attack is apt to initiate this change, hence we see the chronic ostitis in the walls about the nasal sinuses, the middle ear and mastoid. Such a process may go on to complete obliteration of these cavities as one often sees in a cadaver. The phenomenon in the living, is usually seen in the mastoid process when doing a Radical Mastoid. In their clinical aspects one can differentiate the cases of primary osteomyelitis from an ordinary sinusitis by the greater severity of the symptoms—both local and general.

In January 1921, a baby nine months old was brought to the clinic with a fistula about half an inch below the lower eyelid, in about the region of the infra-orbital foramen. It had had a very acute illness a few weeks before this with great swelling of the face. The swelling had softened and was opened. On probing the fistula, an area of bare bone was felt which seemed to be already loose. I opened the fistula, got an exposure, and removed a small sequestrum, but the wound did not heal. It was dressed daily, and any further operation was postponed to give Nature a chance to separate whatever portion of bone had undergone necrosis. At the end of six weeks the wound was opened and enlarged outward over the molar bone. The necrosis was found to be quite extensive, and the whole of the molar bone came away as a sequestrum. The antrum was of course involved, but gave no further trouble after the dead bone was removed, and the wound healed up quickly.

On February 4, 1921. I was called to see a baby nineteen months old, who had become suddenly ill in the previous 24 hours, with high temperature—(104 to 105)—and rapid swelling of the left side of its face. The swelling of the face was so extreme, that both eyes were closed, and the child was a sad sight. I took it to the hospital immediately and under anesthesia made an opening under the cheek similar to the incision in the Caldwell-Luc operation. There was a considerable amount of pus, and an opening on the anterior part of the bone communicated with the antrum. The antrum itself was a small cavity underneath the floor of the orbit. An openinng was made from this cavity into the nose. The opening above the gums was packed lightly with gauze (Iodoform). Recovery from the local condition progressed rapidly, but the child developed a lung abscess on the right side. About two weeks after the operation, during a fit of coughing, a sudden swelling occurred between the fourth and fifth ribs on the outside of the chest. I opened this, and evacuated a half pint of pus, leaving drainage in the cavity. Still the temperature ranged every day around 105°, and we found from X-ray and physical examination that an abscess had developed in the left lung. During a coughing spell one morning while I was in the room, the child began to choke. I picked it up by the heels, and a half

pint of pus ran out of its mouth. Still no change in the temperature. Nearly a week later, the nurse told me there seemed to be a little swelling in the lumbar region. On looking at the lower part of the child's back this was quite evident. I gave it an anesthetic, and by deep incision I tapped an abscess which must have contained 8 or 10 ounces of pus. It now started to get well, and made a quick recovery though at times there appeared a little swelling in the cheek, and a little pus exuded from above the second bicuspid tooth. After nine months this tooth was removed, and a small sequestrum came with it. It was about the size of a bean and was held in situ by the root of the tooth. It then apparently healed up, but just lately I have found another small fistula over the lateral incisor and can feel a piece of bare bone at the bottom. This will have to be removed although it does not seem to be causing any symptoms.

In June 1922, I was called to see a boy 12 years old who had been ill five days previously with great swelling over his right frontal region, which rapidly extended over the whole of the scalp. On the second day a specialist made an opening over the frontal, and found sero-pus under the periosteum. He went through the bone into the sinus, and found the mucous membrane lifted off from the bone with a collection of sero-pus between the membrane and the bone. A drainage tube was inserted and no attempt was made to open other sinuses. A consultant, called from New York, had advised opening the other sinuses on that side. When I saw the boy, he was greatly swollen all over his scalp with lobular swellings limited by the suture lines. He was extremely ill—temperature 105°, but no signs of meingeal irritation. There was no fluctuation in the swellings, and I advised waiting till that could be determined before operating. Two days later I saw him with Dr. Elsberg of New York. He had then marked fluctuation in the swellings, and signs of meningeal irritation. Koenig's sign was positive—retraction of the head, severe pain, and great irritability.

The swellings were opened, and a considerable amount of thick pus was found in each one with bare bone at the bottom of the cavity. He made a rapid recovery from the acute condition, but has had numerous fistula. Dr. Elsberg has removed numerous sequestra since then, some of which involved the whole thickness of the bone. His absolute recovery now seems quite assured. This case I think was primarily osteomyelitis, and the lining of the frontal was involved the same as the external periosteum.

DR. MAYER asked if Dr. Faulkner thought the term myelitis a proper one in these cases where there was no medulla; would it not be better to say osteitis?

He brought up that question, for he was again reminiscent. He was the first one in America to call attention to accessory sinus disease in children, as Dr. Dean had remarked recently. At that time there was a statement made by someone that there was no such thing as accessory sinus disease in children—that it was a tuberculosis.

DR. HURD said that Dr. Dan Mackenzie had published the best paper on osteomyelitis that he had ever read on the subject. Dr. Ross's second case which he seemed to regard as cured, would probably light up again after a while. These cases offer an almost hopeless proposition excepting those of isolated structure; but in ordinary osteomyelitis the prognosis is extremely bad.

Referring to sinus disease in children, Dr. Hurd said he had recently seen a child a month old with an antrum that held two drams of pus, and the whole face badly swollen. The child was operated and two days later died of thrombo-phlebitis. It must have been an osteitis.

DR. KELLEY: Dr. Byrd had a case at the Manhattan E. E. and Throat of multiple osteomylitic areas on which he operated ten or twelve times. It remained quiet for about a year and he now understood it was flaring up again.

DR. MCCULLAGH said he had seen the case referred to by Dr. Kelley within the last two or three weeks and it was perfectly well then, although it may have flared up again. In his service Dr. Byrd also had a similar case in a child of less than six months of age, which had all the symptoms described by Dr. Faulkner which promptly recovered after external operation and removal of the sequestrum.

DR. VOORHEES said that it seemed to him that in many of these cases of osteomyelitis and osteitis there was an imbalance of the endocrine system; therefore when vaccines were used, they failed to give results, either the outogenous, or the stock vaccines. It would be interesting if some one would try the endocrine treatment in association with vaccine therapy and see whether that would increase the patient's resistance. Unless that could be done in some way, all local work would go for nothing, since most cases of multiple or diffuse osteitis relapse from time to time.

DR. LEDERMAN referred to a paper read before the Section by Dr. Callison in reference to a retardation of bone production in some cases where the mastoid wound would not heal, and thyroid treatment was tried with good effect.

DR. FORBES said there must be a type of case with rapid destruction of bone as well as the slow procedure with exfoliation, and cited a case seen with Dr. Sheehan. It was a question what would be the ultimate result. It was a rapid process in which the original foci was in the frontal bone, secondarily in the parietal and finally back to the occiput, with a tremendous surface destruction of bone. The process was acute, the patient died. He did not know just the type of infection present in this case.

DR. FAULKNER, replying to an inquiry, said that a blood culture had been taken and was negative.

He emprasized what Dr. Mayer had said:—that there was no medulla, no bone marrow in these cases, hence osteitis is the better term.

The prognosis in these cases is not so bad as Dr. Hurd seems to think. If you keep at them till all the dead bone is removed, they will heal up perfectly well. Unless the long suppuration produces amyloid disease there is nothing in the local disease to kill them. I know of two cases which have been entirely well for two years.

The Use of Acetysalicylic Acid in Advanced Tuberculosis of the Larynx.
Dr. Joseph D. Kelley.

A little over two years ago I began to use Acetysalicylic Acid in Advanced Tuberculosis of the larynx for the relief of pain and distress on swallowing.

Reasoning on the knowledge of the effectiveness of this drug in the post-operative treatment of tonsillectomies where we know it is most helpful in alleviating the pain and soreness during the first few days of convalescence, I concluded that it might be of service if any throat condition where we were dealing with an inflamed, granulating or ulcerated membrane. Such is the condition that we find in advanced tuberculosis of the larynx, and having a case under treatment at this time which presented the advanced picture, I used acetylsalicylic acid (20 grs.) suspended in one-half glass of water as a gargle. This was tried in the office one morning and the patient detained to watch the effect. To my surprise and delight when he was ushered into the treatment room after remaining in the waiting room for fifteen minutes after using the gargle, the patient was so pleased with the effect himself that he demonstrated to me the fact that he could swallow without distorting his face in agony and lifting his shoulders in an attempt to relieve the conclusion on the act of deglutition. Needness to say I immediately prescribed twenty chartulae each containing twenty grains of acetylsalicylic acid with the instructions to put one powder in one-half glass of water—stir thoroughly so as to obtain as complete a suspension as possible, and use as a gargle fifteen minutes before meals.

This patient returned to my office one week later and informed me that he was delighted with the powders and gave me a list of all he had eaten for breakfast that morning. He also advised me that he was able to rest easier and sleep longer than he had at any time for the month prior to his last visit. This patient was typical of all T.B. cases, he was hopeful and was sure now that he was able to eat and sleep better it was only a matter of time until he would again be able to return to work. He didn't return to work—he did live for three months during which time he was relieved by the use of the gargle.

The next case in which I used this treatment, which is the second case of a series of four which I shall mention, was that of a woman thirty-four years old, who had been under the care of Dr. Feinberg of this city. This patient had been in bed for two weeks, during which time Dr. Feinberg had used everything within reason to relieve her, but without much avail. Her appeal was, that if she could eat and gain sufficient strength to get on a ship bound for Germany, she was sure she knew of a place where she could get well. She was relieved by the use of acetylsalicylic acid gargle, made the trip to Europe and lived for two months after her arrival.

The third case was that of a young man 29 years old who was under treatment at St. Gabriel's Sanatarium and was brought home by his family because of his desperate condition and his inability to swallow without severe pain and agony. He lived but six weeks after coming to my office, but was relieved and made fairly comfortable during that time.

The fourth case was one of a man 39 years old who was referred to me by Dr. Thomas Brennan of Brooklyn. This man was a known T.B. and had been living in a small town in New Jersey for two years because the doctors had advised him to leave the city. Things went well with him until his interest in local politics got the best of his good judgment and his orating produced a sore throat. He came to my office with the same appeal—"If I can only get some food into me and get some strength I am sure I shall be all right." After his first visit he informed me that he found, that if he swallowed some of the gargle he was more relieved. This man came to my office for nearly two months after I first saw him, but during the last month of his life was confined to bed.

As I have stated above in advanced cases of T.B. laryngitis acetysalicylic acid will relieve odynophagia. However, I have not found it to be of any value in those cases without odynophagia and it has no curative properties. Why it can be used successfully in the form of a gargle in these cases, I think, can be explained by the fact that most of the ulceration is found about the epiglottis, the pharyngeal surface of the arytenoids and aryten-epiglottic folds, those surfaces which come in contact with food in the act of deglutition. Also, in the advanced cases the pyriform sinus on either side becomes a reservoir for fluids, so that we may find milk in these sinuses an hour after the patient has had a glass of milk to drink. This I believe is a factor to be considered in using this medication in suspension. If insufflation is used the patient is caused considerable distress in coughing and the result is not as pleasant or effective. It may be administered by giving the patient a tablet to dissolve in his mouth.

I have been interested to know just which component of the acetylsalicylic compound is responsible for the anesthetizing effect and after consulting several pharmaceutical chemists I find that the acetyl component is small and the combination in itself is very unstable. Those chemists to whom I spoke were convinced that the salicylic component was the active part.

I am aware of the fact that there are other drugs on the market which can be used in this condition but these are either very expensive, at times hard to procure, or administered with a laryngeal syringe or

applicator. Acetylsalicyclic acid is easily procured, inexpensive, simple of administration, and at times will relieve the patient when other drugs fail.

DISCUSSION.

DR. FREUDENTHAL agreed with Dr. Kelley that any remedy should be welcomed that offered any help in these advanced cases of laryngeal tuberculosis. He himself uses orthoform in combination with chaulmestral, a brand of the Chaulmoogra oil; he has also used antypyrin, applying it locally; aspirin works in a similar way, and there was no reason why it should not be good in these cases. He certainly intended to try it, for something must be done to relieve these cases; it was not quite clear to him, however, how the arytenoids could be reached by gargling. He had been surprised to read in the Transaction of the Trilogical Society, a paper by a colleague in Denver who recommended gastrostomy. Dr. Freudenthal did not feel inclined to try such a measure.

DR. M. C. MEYERSON wished to corroborate the value of the remedy from his own experience. Dr. Arrowsmith had sent him out to see an advanced case of tuberculosis of the larynx. Dr. Myerson said he learned tht history of the case and that practically everything had been used including anesthetics, orthoform, cocain, etc., and thinking as Dr. Kelley did of aspirin being used in post-operative tonsillectomy wounds, he suggested using 30 grains to a glass of water. His improvement was phenomenal; the patient said the aspirin seemed to cut the mucus out of his throat.

At the King's County Hospital they saw many laryngeal tuberculosis cases. Some of them he treated with aspirin and it worked in every case where there was ulceration. To prove that the salicylic acid ?????? is responsible, Dr. Myerson said that in the past four months he had been using sodium salycilate not only for post-operative tonsillectomy wounds but also for laryngeal cases and found it very efficacious.

DR. SHEEHAN said that he has been using aspirin in all sorts of ulcerative conditions of the mouth, throat and larynx with gratifying results for the past six years. Mr. Harmer of London told me some six years ago of its marked analgesic properties and combined with orthoform he said made one of the best quietors in ulcerative conditions of the throat he had yet met with.

The formula which Dr. Sheehan uses at the Post-Graduate Hospital is: R/orthoform, 8; aspirin, 4; pulv. acaciae, 8; sacch. lactis qs. ad, 30.

Insufflation of the powder several times daily works admirably in all sorts of ulcerations of the mouth, throat larynx etc. It is particularly serviceable in tuberculosis of the larynx and after tonsillectomies.

It does not very well dissolve in water and is readily decomposed on boiling. It is marked analgesic, detoxicator, and somewhat of an antiseptic.

DR. LEDERMAN said that several years ago he had directed attention to an excellent application in these painful and distressing ulcerations in tubercular laryngitis, called Cresatin (Sulzberger). It is a metacresol compound, possessing the characteristic antiseptic and analgesic properties of the cresols without their objectionable features. It is the least toxic of this group and is devoid of irritating or escharotic action. Where the parts are very sensitive, a previous application of cocain should be used, but Dr. Lederman had frequently applied cresatin in full strength without any cocainization of the parts. It is very effective in any ulcerative lesion about the mouth and he has applied it with excellent results in furunculosis of the external auditory canal. Dr. Cyril Barnet of this city has called attention to its beneficial properties and prompt action in gonorrheal ophthalmia.

DR. UNGER cautioned against the use of the term aspirin on prescriptions as a number of concerns are bringing out tablets called aspirin, but some do not contain more aspirin than talcum does. The term acetylealicylic

acid should be used, and also because it is cheaper than anything under the name of aspirin.

DR. KELLEY, replying to Dr. Dougherty, said he would like to have him try acetylsalicylic acid gargle on some of his advanced cases of laryngeal tuberculosis with ulceration on the arytenoids.

The Scialytic Light. Dr. Samuel McCullagh.

To be published in a subsequent issue of THE LARYNGOSCOPE.

SECTION ON OTOLOGY.

April 13, 1923.

Unilateral Nerve Deafness. Dr. A. Palmer.

On August 7, 1922, S. L., a driver, was thrown from his wagon and injured his head and both arms. He was taken to St. Mary's Hospital where he remained unconscious 7 days and then gradually recovered. There was bleeding from the left ear and X-ray of skull showed a fracture of left temporal occipital region. On October 28, 1922, one month after, was discharged from the hospital. He was referred to me for otological examination, from the point of view of industrial insurance compensation. At this time he complained of whistling noises and discharge in the left ear, and attacks of dizziness on arising. He stated that previous to the accident he had had no signs of ear trouble and did not complain at the time of this examination of any impairment of hearing.

Physical Examination. The patient was well built and nourished with marked paresis of the muscles supplied by the left facial nerve The mental reactions were sluggish.

Otological Examination. Discharge which appears to be dried excretions from the middle ear mixed with cerumen was present in the left auditory canal. Left drum was thickened and all normal land-marks were absent or diminished. There is dull redness in Schrapnell's membrane. Oscillation of drum on vibration was diminished. A small perforation was present.

Functional Examination. October 28, 1923. Right ear, normal. Left ear: watch, 0; whisper, 0; spoken voice, close to ear only; acoutmeter, bone conduction only; Rinne, negative; Schwabach, decreased; tuning forks, hears only C.3 and C.4 by air conduction only, all by bone conduction; Galton whistle, slightly lowered.

Test of Vestibular Apparatus. Spontaneous nystagmus, not marked in any direction; vertigo, present; Romberg, slight swaying.

Turning Tests. Turning to the right with head upright position, nystagmus to the left 4 sec.; turning to the left with head upright position, nystagmus to right 14 secs.; turning to the right with head 120′ forward, no response; turning to the left with head 120′ forward, no response.

Coloric. 68′, no nystagmus either ear after 3 min.; 105′, nystagmus 10 secs. right ear. Nor. left. No nausea. Past-pointing, sub-normal.

Patient was seen again February 20, 1923. He still complained of dizziness and tinnitis left ear. Function of muscles supply by left facial nerve much improved.

Functional Examination. Hearing test right ear, normal; Hearing test left ear: watch and whisper, 0; voice, 12 feet.; acoutmeter, bone only; tuning forks, C.3, C.4 are only forks heard by air conduction; Schwabach, diminished; Weber, not well lateralized; Galton whistle, normal.

Vestibular Tests. Fistula tests, no response; spontaneous nystagmus, none in any direction; vertigo, present; past-pointing, sub-normal; Romberg, slight swaying.

Turning Tests. Reaction similar to previous test.

Caloni Tests. At 68′, no response left ear in any position after 3 min; at right ear failure of virtual response but nystagmus 5 sec. on stimulating horizontal canal.

April 7, 1923, functional examination of ear, practically the same as in February, 1923. The ear drum appears more nearly normal. Vibration of the lower portion is good. There is still dull redness of Shrapnell's membrane.

Conclusions. At time of first examination:

Diagnosis. Fracture of vault of the skull extending into petrous portion of the left temporal bone with injury to the fibres to the left facial and auditory nerve. Function of the cochlea and vestibular branches of left auditory nerve, markedly diminished.

Prognosis. Hearing on the left side will probably be impaired permanently. Disability for left ear at present, 90 per cent. There is a possibility that this disability will diminish to 50 per cent in the course of one year after which time there will probably be no improvement.

Comment. This case is presented in the hope that it may prove interesting from the point of view of industrial insurance. My problem was to be fair to both the patient and the insurance companny. I agree with Dr. Fletcher that more thorough examination, deeper thought, and more careful analysis of industrial injuries involving ears and more comprehensive reports to the insurance companies should be the physician's endeavor.

Reference. "Determination of Disability as to Loss of Hearing and the Importance of Vertigo in Industrial Accident Cases," by Dr. Harold A. Fletcher, of San Francisco at the meeting of the A. M. A., May, 1922.

DISCUSSION.

DR. MARK GOTTLIEB said that cases of this kind were very interesting and had certain very definite practical lessons for otologists who are doing work in oto-neurology, and those treating persons who expect to receive compensation from various compensation commissions. He had been much impressed by what Dr. Palmer said about the hearing conversation at 12 feet and the hearing of the C.2, C.3, C.4 forks by air conduction only, the hearing with the acoutmeter over the mastoid bone, and the fact that the patient gave no responses at all to stimulation of the static-kinetic labyrinth on that side. Did Dr. Palmer use the noise apparatus when he tested the patient's hearing for conversation?

Did he douche the unaffected ear with water at body temperature when the diseased ear was being tested with the C.2, C.3, and C.4 tuning forks and the Galton whistle?

From the facts presented it did not seem logical to assume that the patient had any hearing on the diseased side at all, for there was no response to caloric stimulation on douching.

DR. MAYBAUM said that some years ago, at the Manhattan Eye, Ear and Throat Hospital, he had conducted an investigation in Dr. Beren's service of all cases of impaired hearing with or without a history of vertigo. Quite a large number gave histories of head injury at some time or another. Some had total loss of function, both cochlear and vestibular. Some had no change whatever in the vestibular reactions, but different degrees of impaired hearing pointing to changes in the perceptive apparatus; cases of recent injury showed spontaneous signs—nystagmus and deviation from normal pointing.

(To be Continued)